

Volume I

URGENT SURGERY

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Edited by

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244 FIGURES HAVING 572 ILLUSTRATIONS

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to

VOLUME I

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Preface

The scope of emergency surgery, or rather urgent surgery, has within the past few years become so wide that most monographs on this subject published only a decade ago have become inadequate.

It should be borne in mind that some years ago what was referred to as emergency surgery was limited to accidents principally traumatic in nature with the addition of threatened suffocation through the lodgment of a foreign body in the trachea. Monographs on this type of surgery were by no means prepared for experienced surgeons but rather as a guide for general practitioners thrown upon their own resources at places far from hospital facilities. Gradually, with the development of diagnostic technics, there was added an important chapter on the rupture of such viscera as the appendix, the gall bladder, the stomach, the duodenum and the fallopian tube. Intestinal obstruction, too, was included as a condition calling for emergency intervention by surgical operation.

Subsequently, many problems of importance in the earlier days of emergency surgery had to be reevaluated, because the elements of time and space were overcome by the extensive development of transportation. Today most rural districts command the facilities afforded by well-equipped hospitals staffed by competent surgeons within comparatively easy reach, so that procrastination in conditions requiring prompt operation is no longer excusable. This factor alone justifies the abolition of the term *emergency surgery* and the substitution of *urgent surgery*, which designation we have adopted for this book. In other words, while formerly the word "emergency" implied the necessity for intervention within minutes or hours, we must now take into consideration such cases in which operation becomes imperative within a few days, if life or health is to be preserved.

We hardly deem it necessary to apologize for the introduction of brief historical reviews in some of the sections of this book in order to elucidate the development of our knowledge of the problems under consideration. Discussions of surgical anatomy are regarded as essential, because, in the type of cases requiring immediate or almost immediate intervention, the normal anatomic landmarks are often distorted to an even greater degree than is encountered in elective operations. This fact suggested the usefulness of comparing the operative technic under more or less controlled situations, that is to say, under more or less familiar pathologic conditions, with the technic to be employed under urgencies. It is self-evident that one mastering the technic of elective surgery will not find himself lost when confronted by the deviations and aberrations incident to conditions of an urgent nature.

The present work has been prepared by surgeons recognized as authoritative in their respective fields, both in the United States and abroad.

It is with deep regret that we announce that three of our collaborators have died: Professor Bosh Arana of Buenos Aires, Dr. George de Tarnowsky of the University of Illinois, and Dr. Joseph Beck of the University of Illinois. The chapters contributed by these three distinguished specialistic surgeons have, however, been brought up to date.

The illustrations are mostly original ones, and have been executed in this country under the able supervision of Mr. Tom Jones, Professor of Medical Illustration at the University of Illinois. To him, as to his associates, Miss B. Abrahamson, Miss R. Coleman, Miss J. Lorenz, Miss M. McConnel and Miss J. Morgan we express our sincere indebtedness for their artistry. Some of the illustrations have been provided by Messrs. M. Landsberg, M. Szabo and Hunter to whom thanks are due.

Our appreciation is also expressed to Miss E. Salmonsens of the John Crerar Library, for the preparation of the subject and the authors' indices.

Acknowledgment is due the distinguished contributors for the painstaking preparation of their various chapters. Indebtedness is expressed to my good friend and colleague, Brigadier General Gustavus M. Blech, for many suggestions. Last but not least, I gratefully acknowledge the helpful cooperation of Mr. Charles C Thomas, the publisher, during the preparation of the work.

JULIUS L. SPIVACK

Chicago, Illinois
May, 1946

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Volume I

URGENT SURGERY

Chapter I

Intravenous Infusion. Blood Transfusion

By JULIUS L. SPIVACK

INTRAVENOUS INFUSION (VENOCLYSIS)

DEFINITION. The term venoclysis is derived from the Latin word *vena*, a vein, and the Greek word *κλυσις*, an injection, and signifies the introduction of fluid into a vein. If the amount introduced is small, it is spoken of as an injection for which usually a syringe is used. If the amount is greater than contained in a syringe, we speak of an infusion. In the latter case the fluid is allowed to run into the vein by force of gravity.

HISTORICAL. The idea of introducing drugs into the veins came about the same time as the idea of blood transfusion; indeed, in many cases the same men who were interested in one became pioneers in the other. Christopher Wren, of Oxford, the famous architect of St. Paul's Cathedral in London, was probably the first who, in 1656, injected drugs into the veins of dogs. The first injection of drugs into the vein of a man was done by the French physician Colladon in 1657. Owing to supposed hazards connected with such an undertaking, he tried it on a criminal condemned to be hanged. Wren, in 1662, injected vinum emeticum into the vein of a man. Timothy Clark, in 1663, presented a paper before the Royal Society in London on the subject of intravenous injection of drugs into animals. Among the drugs used were emetics, cathartics, diuretics, cardiacs and opiates; the results were rather inconclusive. He, therefore, doubted the usefulness of intravenous medication. Some untoward accidents occurred, such as fever, infection and emboli and the method fell into disrepute and was used only sporadically in the seventeenth and eighteenth centuries. Interest in this type of treatment was revived in the nineteenth century, when Latta, a Scotch physician, in 1831, introduced intravenous infusion of saline solution for the treatment of dehydration in Asiatic cholera. After that intravenous infusion of saline solution was more frequently though not extensively used. It was only during World War I (1914-1918) that many fluids were extensively administered intravenously, mainly to combat shock.

GENERAL CONSIDERATIONS. Introduction of fluids into veins is carried out for different purposes. It may be employed for the administration of drugs, which if introduced by other routes would either produce pain (if injected subcutaneously or intramuscularly) or lack a quick action (if introduced per mouth). In other cases, intravenous injections are used for diagnostic purposes, such as the determination of the function of the gallbladder, liver or kidneys. In a great number of cases a small amount of fluid is injected into a vein for a local, sclerosing action, as in the treatment of varicose veins. The greatest importance of intravenous infusion in surgery lies in its nutritional value. It is employed as a pre-

operative and postoperative measure and also to prevent and combat shock.

INDICATIONS. Several objectives can be attained by employing intravenous therapy: (1) Prevention of dehydration in the days after operation when the intake of fluids is greatly diminished and the expenditure is increased due to fever which often follows any surgical procedure. (2) Introduction of a sufficient number of calories to provide bodily energy. (3) Replacement of organic or inorganic ingredients in the blood or tissues lost in some pathologic conditions, as sodium chlorides in the blood during intestinal obstruction, glycogen in the liver or in the muscles in wasting diseases. (4) As a therapeutic measure by introducing hypertonic solutions in conditions in which dehydration of tissues is aimed at, for example, in cases of edema of the brain, or to increase the function of the kidneys in anuria or oliguria.

Dehydration is probably the most frequent indication for the intravenous infusion of fluids. It occurs through abstaining from fluids, through excessive loss of water as the result of persistent vomiting, diarrhea, increased sweating, or excretion of large amounts of urine as observed in prostatic obstruction. Under normal conditions an adult of average size loses about 2,400 cc. of water daily. This takes place mainly through the excretion of urine in which about 1,500 cc. is lost, through perspiration in which about 450 cc. is lost, through respiration in which about 300 cc. is lost, and through the evacuation of feces which causes a loss of 150 cc. Whichever the cause of dehydration, it affects the body in several ways. There is a loss of weight incurred through the reduction of tissue fluids and disturbance of the acid base equilibrium which usually leads to acidosis. This latter is chiefly due to slowing of renal function with reduced excretion of urine and retention of acids. Another effect is an increase in non-protein nitrogen in the blood and elevation of the temperature as a result of the diminution of circulating fluids. For all these reasons the correction of dehydration is imperative.

SOLUTIONS USED IN INTRAVENOUS INFUSIONS. Two kinds of fluids are used in the vast majority of cases, namely, normal salt solution and glucose dissolved either in distilled water or in normal salt solution. While in former years normal salt solution was used more often than glucose, this is not the case now. As a matter of fact, there is probably a preponderance of the employment of glucose over that of normal salt solution.

Normal salt solution contains about 8.5 gm. of sodium chloride in 1,000 cc. of distilled water. As the normal intake of sodium chloride should range between 6 and 10 gm. daily, infusion of 1,000 cc. of normal salt solution will satisfy the body's daily needs. If a larger amount of normal salt solution is given, an excessive quantity of sodium chloride

tissues; the increased amount of salt will draw fluid, thus producing edema of the tissues. Under ordinary circumstances when we give 2,000 to 3,000 cc. of normal salt solution daily as a postoperative measure for two or three days, one hardly will observe edema; but if normal salt is given for a longer period, edema will occur. This usually is observed on the back and not in the extremities. If that takes place, administration of normal salt should be replaced by injection of 5 per cent glucose dissolved in distilled water because it effectively eliminates the retained fluids from the tissues. Normal salt solution is specifically indicated in illnesses in which sodium chloride is lost, as in high intestinal obstruction, and in other disorders characterized by prolonged vomiting. It is also advisable to give salt solution in preference to glucose in case of dehydration because it retains the fluids in the tissues.

Solution of Glucose. Solutions of glucose are used in two strengths—5 and 10 per cent. Some use the former strength for subcutaneous and the latter for intravenous injections. In either strength glucose yields definite caloric values and does not produce edema of the tissues, even if it is introduced in great quantities. The importance of employing glucose solution lies not only in its food value but also in its ability to prevent ketosis. It must be remembered that when the intake of food is diminished, energy is derived from the decomposition of glycogen; and when this ceases, the fat becomes the source of energy and when fat is incompletely oxidized, ketosis ensues. Glucose prevents the rapid utilization of the stored glycogen and thereby also ketosis.

Hypertonic solutions of glucose (from 25 to 50 per cent) have only a limited application. They are used in some cases of anuria or oliguria to draw the fluids from the edematous tissues into the blood stream with the aim of being eliminated by the kidneys.

Acacia. This is usually given in normal salt solution at a strength of 6 per cent. The amount given in one sitting is about 500 to 600 cc. Owing to its osmotic pressure, acacia has a tendency to stay in the blood vessels, thus retaining the fluid volume and the blood pressure, while glucose or normal salt solution injected into the veins leaves them rapidly. Therefore, in shock it is by far more efficacious than glucose or normal salt solutions. However, acacia is inferior to plasma or blood; and since plasma is a simple and safe procedure, acacia is not used much now. It may retain its place only for two reasons: it is much cheaper than plasma and the latter may not always be available. There is, however, one group of conditions in which the usefulness of acacia is great and that is in hypoproteinemia with severe edema. Acacia is a good diuretic and diminishes the edema.

Sodium bicarbonate in 5 per cent solution is given to combat acidosis occurring in diabetes, renal insufficiency or occasionally after the administration of large doses of sulfa drugs. The amount given varies from

300 to 500 cc. One has to bear in mind that the injection of even one dose of sodium bicarbonate solution may change acidosis into alkalosis (as can be ascertained by determining the carbon dioxide combining power which normally ranges from 55 to 80 cc. per 100 cc.). Therefore, a second injection of sodium bicarbonate must never be given until the combining power of carbon dioxide in the blood has been determined.

Hartmann's solution is occasionally given in case of acidosis. It acts analogously to sodium bicarbonate solution but more slowly. Ordinarily in acidosis quick action is desirable, therefore, sodium bicarbonate is preferable.

THE AMOUNT OF FLUID INFUSED varies, depending upon the degree of dehydration, the amount of food taken postoperatively by mouth, the temperature and the humidity of the room, whether the patient has fever, the character of the disease, etc. However, in ordinary cases when fluid is given as a postoperative measure, 2,000 cc. of normal solution or glucose is sufficient. However, in order to be more exact in balancing the intake and the output, one has to multiply the volume of excreted urine for twenty-four hours by one and four-fifths and give such amount of fluid intravenously.

THE RATE OF INJECTION should be 50 to 75 drops per minute. As fluid usually leaves the vascular tree very soon, such an amount is safe and does not overload the heart. If the patient has myocardial damage, the rate should be watched; if the patient has substernal pain, a sensation of pressure in the chest or dyspnea, the infusion should be discontinued at once and resumed a few hours later at half the speed given before.

CONTRAINDICATIONS. *Contraindications* are very few. Continuous intravenous infusion is contraindicated in pulmonary congestion, failing heart, as manifested by a fast and irregular pulse and drop of the blood pressure, and in marked hypertension.

DANGERS AND COMPLICATIONS. Among the dangers of continuous infusion is the possibility of overloading the heart by too rapid injection. The infusion rate of 300 cc. per hour is safe because ordinarily the solution, particularly saline, leaves the vascular tree soon after it is injected. If the patient has a feeling of oppression around the chest or dyspnea, the injection should be discontinued. There may arise few accidents due to faulty technic, such as breaking of the needle, entering the artery instead of the vein, air embolism, injuring a nearby nerve trunk; however, these complications are very infrequent. The entering of an artery instead of a vein is a more frequent occurrence than is commonly believed. Fortunately, no ill results occur; however, a few cases of arterial aneurysm or of arteriovenous aneurysm caused by this mishap have been reported. One has to be particularly careful not to enter an artery instead of a vein when using sclerosing solutions; there are reports of

occluding the artery with ensuing gangrene, when a sclerosing solution was injected into the artery instead of the vein. The most frequent complication is a pyrogenic action of a solution. Immediately after it is injected the patient's temperature rises and the pulse becomes accelerated. In most of the cases this is due either to contaminated fluid or to the tubings, which probably were not clear of particles from previous injections. Sterilization alone cannot prevent such a reaction; it cannot be too strongly emphasized that all tubes be cleansed of every foreign particle remaining after each infusion. In order to overcome this objection, tubes consisting of a flexible, transparent, plastic substance which is discarded after each injection have appeared on the market. This eliminates the most frequent cause of pyrogenic reactions.

SURGICAL TECHNIC. The armamentarium for infusion consists of a funnel containing fluid and tube connected with a needle, which is inserted into a vein. This is the simplest type of equipment (Fig. 1). There are more complicated types supplied with vacuum container in which the fluid is protected against contamination. Vacuum containers are by far superior to the simple type, because the solution is ready for use, sterile, perfectly protected against contamination, simple to keep in storage and to transport. At first glance it may seem that they are more difficult to handle, but after becoming familiar with them the technic is very simple. We use the infusion apparatus, manufactured by Baxter Laboratories, Glenview, Ill.

Although the accompanying illustrations are clear and the legends self-explanatory, it will not be amiss to give a brief description of the apparatus. It consists of a sealed jar, which contains sterile, pyrogen-free normal salt or glucose solution, vacodrip, rubber tubing, and adapter for the needle and a needle. The sealed jar containing the fluid is known as vacoliter and is made in different sizes (Fig. 2). The fluid in the jar is under negative pressure, hence the name vacoliter. The jar is covered by a metal disc on which is written the name of the solution in the vacoliter (Fig. 3); this cover also serves as a tamper-proof seal. This tamper-proof disc is broken by pulling the corrugated tab straight out and down (Fig. 4). Next, the outer cap is removed (Fig. 5) and the metal identification disc is lifted (Fig. 6) exposing the rubber disc

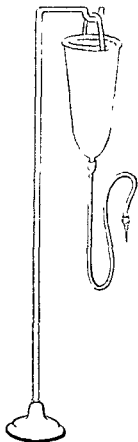


FIG. 1. Simple container for venous infusion.

(Fig. 7). Two depressions are now seen in this thin rubber disc. This indicates that the vacuum is intact. If the depressions are absent, it indicates that the vacuum has been broken and the solution should not be used. Thus we see that the vacuum is used not only in order to make possible the sterilization of the contents without breaking the glass but it serves



FIG. 2. Different sizes of vacoliter.

(Courtesy of Baxter Laboratories, Glenview, Illinois)

as a tamper-proof device. Next, the vacoliter is turned upside down for a moment (Fig. 8). This is done to moisten the hole in the stopper in order to facilitate the insertion of the vacodrip (that is the cylindrical glass tube, which serves as a part of the connecting tubing) into one of the holes of the stopper. Now the rubber disc is removed (Fig. 9) and the stopper is thus exposed with two holes in it. A glass air tube passes through one of these holes; the other is the "free hole." Next, the end of the vacodrip is inserted through this "free hole" into the vacoliter

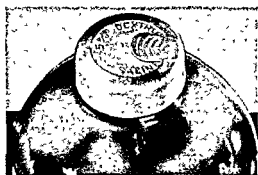


FIG. 3. Tamper-proof seal and identification disc.



FIG. 1. Corrugated tab pulled straight out and down.



FIG. 5. Removal of outer metal protective seal.



FIG. 6. Convenient way to remove metal identification disc.

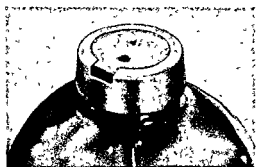


FIG. 7. Depressions in the rubber disc show vacuum is present.

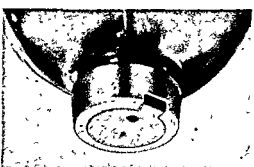


FIG. 8. The vacoliter is turned upside down.



FIG. 9. Removing rubber disc and exposing holes in stopper.

(Fig. 10). After that the shut-off clamp located about six inches below the vacodrip is closed, the vacoliter turned upside down and suspended by means of attached band and bail.

Due to the fact that air is present in the vacodrip no solution, or very little, will escape from the vacoliter into the vacodrip where it will oc-



FIG. 10. Insertion of vacodrip into free hole of stopper.

cupy the lower portion. The tubing is now held above the solution level of the vacodrip. The solution is allowed to run through the vacodrip into the tubing by opening the clamp and immediately closing it tightly. (By momentarily opening the clamp, the air from the vacodrip and the tubing above the clamp is directed into the tubing below the clamp, thus allowing some solution to run from the vacoliter into the vacodrip and tubing.)

The tubing above the clamp is now squeezed, forcing the air bubbles through the lower chamber of the vacodrip into its upper half. The lower portion of the vacodrip is thus filled with the solution while the upper half contains air. This air acts as a cushion, facilitating the regulation of the flow of fluid from the vacoliter into the lower portion of the

vacodrip and tubing after the screw clamp has been adjusted. The tubing end is held above the solution level of the vacodrip and the clamp is slightly opened until all of the tubing and the needle adapter are filled with solution (Fig. 11). The clamp is then closed. The needle is inserted into the vein as outlined below and the adapter is connected with the



FIG. 11. Filling the vacodrip and infusion tubing.
(Courtesy of Baxter Laboratories, Glenview, Illinois)

needle. Some prefer to connect the adapter with the needle before inserting the latter into the vein.

The selection of a vein is limited to a very few places (Fig. 12). The most frequently used is the median cephalic or the median basilic in the cubital fossa (Fig. 13A); occasionally veins on the dorsum of the hand or the radial border of the wrist (Fig. 13B), the superficial temporal and the internal saphenous just anterior to the internal malleolus of the tibia (Fig. 14) are used. This latter is especially useful in children (Fig. 15). The choice is influenced not only by the size and accessibility of the vein but also by the length of time of the infusion. Thus, in an infusion lasting for hours, immobilization of the arm for infusion through the cubital fossa tires the patient, while infusions through a vein in the dorsum of the hand keep the patient comfortable.

long bevel; if it has a long bevel, it may penetrate both walls of the vein. It should be sharp because a blunt needle tears the wall of the vein.

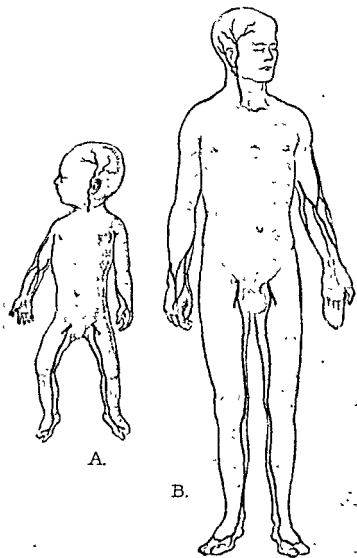


Fig. 12. Superficial veins used for venipuncture.
A. In a child. B. In adult.

The patency of the lumen of the needle should always be tested before venipuncture is made. After a vein has been selected, the cuff of a sphygmomanometer is wrapped around the arm or the leg proximal to the selected vein and the pressure is raised to about 50 to 60 mm. mercury (slightly below the diastolic pressure). The skin is cleansed with an alcohol sponge. Some inject 1 cc. of 1 per cent of novocain solution sub-

cutaneously in order to render the procedure painless; however, we resort to it only in apprehensive patients.

The solution is allowed to flow through the rubber tubing and the needle so as to expel air. As soon as this is done, a clamp is placed on the

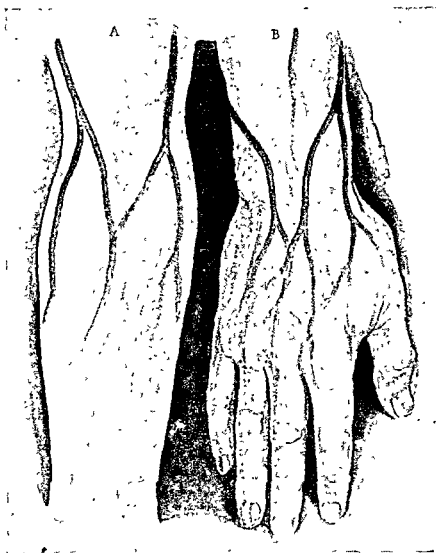


FIG. 13. A. Superficial veins of the cubital fossa used for venipuncture. B. Superficial veins of the dorsal surface of the forearm and hand used for venipuncture.

tube close to the needle. Next, the needle connected with the tube of the infusion apparatus by a glass intermediary which enables one to see whether it entered the vein, pierces the skin only, entering into subcutaneous tissue. It is not advisable to penetrate with the same motion through the skin and the vein as one is liable to pierce both walls of the vein or to enter it tangentially and incompletely. Since it is possible to injure the vein while entering the subcutaneous tissue, it is advisable to

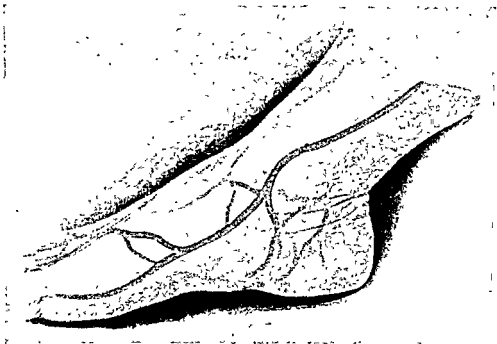


FIG. 14. Internal saphenous vein and its tributaries used for venipuncture

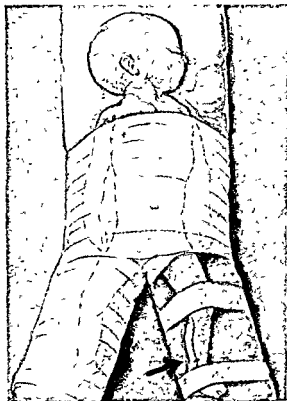


FIG. 15. Internal saphenous vein in a child used for venipuncture

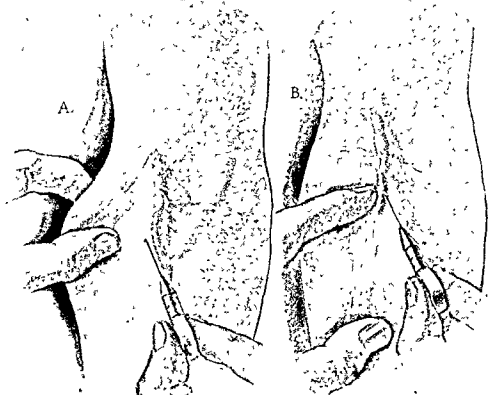


FIG. 16. Technic of venipuncture. A. Drawing the skin lateral from the vein. B. The point of the needle is in front of the vein.

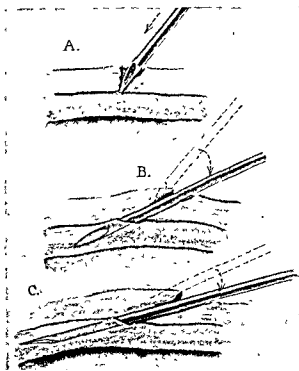


FIG. 17. Successive steps in entering a vein. A. The needle penetrates the skin at 45° . B. The needle enters the vein. C. The needle forms an angle with the vein of about 10° .

move the skin over the vein slightly to the side and then to pierce the skin only (Fig. 16A). After the needle enters the subcutaneous tissue, the skin is allowed to return to its original position, thus bringing the needle in the proper position to enter the vein (Fig. 16, B). The needle is now turned 45 degrees and pierces the anterior wall of the vein (Fig. 17A); as soon as the needle enters the vein, its direction is changed so

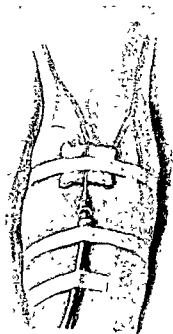


FIG. 18 Method of fixation of the needle and the tube.

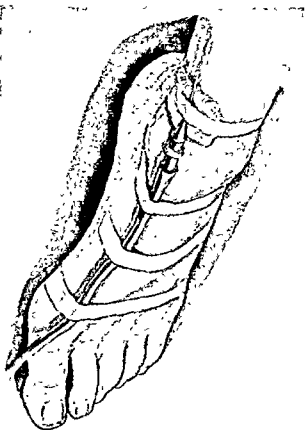


FIG. 19. Method of fixation of the needle and the tube on the foot.

as to form with the vein an angle of 10 degrees (Fig. 17B, C). If the needle is well within the lumen of the vein, blood will run in a forceful stream; if, however, the blood only drops from the needle, the attempt was unsuccessful; the needle should then be withdrawn and reinserted at another point. After the needle is in the vein it should be fastened securely. A convenient method is to place a small pad of gauze between the needle and the skin in order to preserve the 10 degree angle (Fig. 18). This pad with the needle is fastened to the skin with a strip of adhesive; two other pieces of adhesive fasten the tubing to the skin, one close to the needle and the other more proximal (Fig. 18). In a similar manner the needle and tubing are fastened on the foot (Fig. 19).

There are cases when the vein is either too small or not visible because

it is covered with a layer of fat. Shock may collapse the vein so that insertion of the needle becomes extremely difficult. Under such circumstances, one may be forced to expose a vein by dissection and introduce a cannula. The technic is described in the following section on blood transfusion.

BLOOD TRANSFUSION

Blood transfusion is one of the most fascinating chapters in medicine. Being known in a rudimentary stage from remote antiquity it made slow progress in the span of the millenniums, and it was not until the twentieth century that it became a well established procedure for a variety of conditions and also as an important adjuvant in preoperative and postoperative management, thereby contributing to the safety of the most complicated surgical procedures.

HISTORICAL NOTE. Blood has been known as a valuable therapeutic agent since the remote antiquity. According to Pliny, the priests of ancient Egypt considered blood as a rejuvenating remedy and used it in a form of a bath. From them the knowledge passed to the ancient Hebrews, Greeks, and Romans. Pliny also described the drinking of the flowing blood of gladiators in the arena of the Rome circus "as if out of living cups" as a cure for epilepsy. However, even in those remote days there was some vague idea of a blood transfusion; this is evident from the writings of Ovid (43 B.C.-17 A.D.), who in his *Heroides* attributes to Medea, famous sorceress of Colchis, the following: "Why do you hesitate now? Draw the swords and withdraw the old blood and replace the empty veins with the blood of the young."

In order to render blood transfusion a feasible procedure it was necessary to correct several fundamental misconceptions prevalent at that time, such as the origin and course of the blood, and also to learn some of its features, such as coagulation and methods of its prevention and also its immunologic and other biologic properties.

The ancient physicians thought the arteries to be air carrying tubes. However, Aristotle (384-322 B.C.) already considered the arteries to be blood carrying tubes; but he did not know the difference between arteries and veins, considered all of them as tubes carrying blood from the heart where it originated. He knew nothing about the return flow and his conception of it was something like that of a central pool of an irrigating garden, from which water runs to the periphery but never returns to its source.

That the vessels were filled with blood and that their wounding produced hemorrhage was well known in the time of Claudius Galenus (131-201 A.D.) as is seen from his writings:— ". . . arteria quacunq[ue] vulnerata sanguinem egredi videmus" (" . . . we see blood leaving the wounded artery"). Even after it was known that the blood is leaving the heart through the arteries, it was still obscure how the blood reaches from the right to the left chambers of the heart. It was thought for centuries that the blood penetrates through interauricular and interventricular septa and this view prevailed until the sixteenth century. At that time Andreas Vesalius (1514-1564) expressed his doubts as to the correctness of this conception. However, he could not offer any other explanation. This was done by Miguel Servetus¹ (1509-1553), who in 1553, showed that blood enters into the right auricle through both venae cavae, passes

from there to the right ventricle, thence goes through the pulmonary arteries, enters the vascular bed of the lungs, returns to the left auricle through pulmonary veins and from there to the left ventricle. Thus the pulmonary or the lesser circulation was discovered nearly three-quarters of a century before Harvey made his epochal discovery of the great or systemic circulation.

It was left to William Harvey² (1578-1657) to provide the missing link and to show that the blood not only runs from the heart through the arteries to the periphery, which was at that time a common knowledge, but returns from the periphery to the right heart through veins, thus clearly establishing the entire cycle of the circulation.

He made this discovery in 1616, lectured about it since 1619, and published his paper on this subject in 1628.

It is only natural that after the discovery of the circulation the idea should be originated of introducing medicines by the veins, thus giving birth to intravenous medication. And that actually took place immediately after the discovery of Harvey. About the same time more or less persistent attempts of blood transfusion were made.

One of the earliest reports of blood transfusion from man to man is the often quoted case reported by Villari,³ in which he states that the ailing Pope Innocent VIII received, in 1492, blood taken from three boys. All boys died but the blood did not restore the health of the pope who died soon afterward.

A description of a blood transfusion from man to man in which the detailed technic was given is attributed to Andreas Libavius,⁴ a chemist of Halle, who, in 1615, described what we now call direct blood transfusion. His description causes one to think that if it were written in the first decade of the twentieth and not in the seventeenth century:

Let there be a young man, robust, full of spirituous blood, and also an old man, thin, emaciated, his strength exhausted, hardly able to retain his own soul. Let the master have two silver tubes fitting into each other. Let him open the artery of the young man and put into it one of the tubes fastening it in. Let him immediately after open the artery of the old man, and put the female tube into it, and then the tubes being joined together, the hot and spirituous blood of the young man will pour into the old one, as if it were from a fountain of life, and all of his weakness will be dispelled.

Blood transfusion was introduced nearly simultaneously in England and in France. They were mostly transfusions from animal to animal, though in some cases it was from an animal to a human and in only rare cases from a human to a human. At that time it was not much of a replacement therapy. It was done with the hope of changing physical and mental peculiarities of the transfused. The surgeons were interested whether an old man can become young after receiving his blood, whether a ferocious animal will become tame if the former will receive the blood of a tame one, whether husband and wife incompatible in character will straighten their differences if they will be cross transfused, and so on.

In England one of the earliest blood transfusions was performed by Richard Lower⁵ on dogs in 1666, and from a lamb to a man in 1667. Pepys⁶ mentions in his diary one of these experiments.

The transfusions made by Lower produced a great impression in England and they were done later under the auspices of the Royal Society of London.

In France transfusions were started about the same time as in England, the first transfusion on a man being performed by Jean Baptiste Dennis,⁷ in June 1667, who used calf's blood. However, these attempts met with a hostile recep-

tion from the scientific world of France and very soon blood transfusions were forbidden there by law.

In Italy the possibility of blood transfusion was mentioned as early as in 1628 by Johannes Colle, Professor in Padua. Joh. Guilelmus Riva, physician to Pope Clemens IX, in 1667, transfused in three cases blood of a lamb to a man with good results.

In Germany, Andreas Libavius described blood transfusion in 1615.

The complete ignorance pertaining to coagulation, immunology and incompatibility of different blood groups were responsible for severe reactions occurring from time to time. That was the reason why this operation passed into oblivion at the end of the seventeenth century.

The credit for revival of interest in this procedure belongs to James Blundell,¹ an English obstetrician, who has been confronted on several occasions with death of his patients due to postpartum hemorrhage. He suggested, in 1818, to transfuse such patients. Out of ten patients so treated five survived. However, he was confronted with the same difficulties as the surgeons of the seventeenth century, namely, blood coagulation, infections and blood reactions due to agglutination and hemolysis. The works of Dumas, Dieffenbach,¹⁰ and Bischoff¹¹ in the first half of the nineteenth century concerning the prevention of coagulation of the blood by its defibrination removed one of the obstacles. Another method of preventing coagulation consisted in adding different chemicals until, finally, sodium citrate, introduced in 1914 by Hustin^{12,13} of Belgium and by Agote¹⁴ of Buenos Aires, became the safest and most widely used anticoagulant. In the United States, Lewisohn,¹⁵ in 1915, and Weil,¹⁶ in the same year, independently of each other introduced this method. Recently, heparin was introduced as an anticoagulant.

Among other methods preventing coagulation of the blood should be mentioned the procedure of keeping the blood throughout in contact with the intima of the blood vessels. Owing to its technical difficulties this procedure is now discarded. Another method is to leave the blood outside of the blood vessels for a very short time so that there is no time for coagulation to take place. To this group belong multiple syringe method,¹⁷ which may be used in a case of acute emergency, when no more elaborate apparatus is available, and all syringe valve methods.

It was still necessary to overcome the most intricate difficulty, namely, the incompatibility of blood of different donors.

That transfusion of blood was occasionally accompanied by fever, chills and collapse was known already in the seventeenth century. In trying to explain the reason for its occurrence, Cohnheim,¹⁸ in 1882, stated that transfusion of heterogenous blood is dangerous because the serum of the donor may be a direct poison to the corpuscles of the recipient.

Landsteiner,^{19,20} in 1900, made a discovery which should be considered as the greatest single contribution in the development of blood transfusion. He showed the presence of isoagglutinating and isoagglutinable substances in the human blood.

In the following year he demonstrated that the blood of an individual belongs to one of three groups in respect to their agglutinating properties. De-Castello and Sturli,²¹ in 1902, added a fourth group. Jansky,²² in 1907, worked out the reciprocal agglutinating reactions of the four groups and classified them accordingly. Moss,²³ in 1910, working independently, offered his own classification.

These classifications eliminated the greatest danger of blood transfusion and placed it on a safe basis. All that was left to do was to improve the technical aspect, to simplify it and thus to render it available for use by the rank and file of the profession.

INDICATIONS. The indications for blood transfusion are manifold. It is employed as a *replacement* material in hemorrhage so as to increase the volume; it may be employed in hemorrhagic states to increase the oxygen carrying capacity; its employment in case of shock, infectious diseases, intoxications is of great value. It may be used to increase the protein concentration in the blood. For a surgeon its greatest value lies in the preoperative and postoperative management, which enhanced the patient's safety for surgery and conserved many lives that otherwise would not survive operation.

Here is a brief analysis of blood transfusion for each of the above mentioned conditions:

HEMORRHAGE. In hemorrhage, whether due to trauma or to a surgical or obstetrical procedure, such as a ruptured uterus, ruptured tubal pregnancy, premature separation of the placenta, placenta previa, post-partum hemorrhage, transfusion may be a life saving procedure. In hemorrhage from the lungs or of the gastro-intestinal tract, blood transfusion is invaluable because it increases not only the volume of fluid in the vessels but also the oxygen carrying capacity of the blood and combats hypoxemia.

SHOCK. As a prophylactic measure and as active treatment of shock, blood transfusions (or plasma transfusions) are of greatest benefit. Their greatest value is unquestionable not only in case of shock due to hemorrhage, when blood acts as a replacement material, but also in shock of any other origin, such as crushing injuries, burns, excessive manipulations of the viscera in abdominal operations and the like. As the most important mechanical feature of shock, whatever its origin and cause, is the decreased volume of circulating blood, the advisability of adding blood or plasma becomes evident. The sooner this is done, the better the patient's chances for recovery.

In shock caused by burns or wounds there is loss of plasma, and in such cases transfusion of plasma is preferable to whole blood transfusion. The reason for it is that in loss of plasma, the number of erythrocytes in the blood remains the same, but they are suspended in a smaller volume of plasma; therefore, the blood becomes more concentrated and the viscosity of blood is increased, which puts an additional burden upon the heart. Under such a condition blood transfusion will throw additional burden on the heart, whereas plasma or serum transfusion may save the patient's life.

PREOPERATIVE AND POSTOPERATIVE CARE. The value of blood transfusion as a preoperative and a postoperative measure is generally recog-

nized. In clinics in which the cost of blood or plasma is not prohibitive for patients, such as large charity institutions provided with blood and plasma banks for free distribution, or in those treating well-to-do patients, blood transfusion as a preoperative or postoperative measure constitutes nearly one-half of all transfusions. In some clinics blood transfusion is done nearly routinely after every major operation.

INFECTIONS. Blood transfusion is used in some clinics in the course of acute and chronic infectious diseases, such as scarlet fever, undulant fever and subacute bacterial endocarditis. While the results are not as good as in cases in which blood is used as a "replacement therapy" there are many favorable reports to warrant its employment in some cases running a grave course. The improvement obtained is probably due to transfer of normal immunity substances into the blood of the patient whose immunity factors are reduced or impaired. However, for this type of treatment only fresh blood should be used, as stored blood very quickly loses its bactericidal properties.

There are also a number of diseases which ordinarily do not belong to the domain of urgent or emergency surgery in which blood transfusion is useful. We mention it here for the sake of completeness of the indications for blood transfusion. To this group belong different types of blood dyscrasias and cases of debility.

Methods of Blood Transfusion

There are a number of methods used in blood transfusion; most of them now are more of historical interest; others are used today but only infrequently; only a few being widely used at this time.

All methods may be classed into two large groups: I. Direct transfusion, and II. Indirect transfusion.

DIRECT TRANSFUSION. The lumen of the vessel of the donor is connected with that of the recipient either by end-to-end anastomosis or by the intermediary of some kind of a cannula. In either of these methods the intima of the vessels of the donor and of the recipient are in contact so that blood never loses its contact with the intima, thus preventing its coagulation. However, there are several objections to this type of transfusion: (1) It requires great technical skill for its performance. (2) It sacrifices the vein of the donor and of the recipient. (3) There is no way of estimating the amount of blood transfused. (4) It makes transfusion dangerous for the donor if the recipient has an infectious or communicable disease. For all these reasons the direct transfusion is no longer used.

INDIRECT TRANSFUSION. Many variations are possible. One may use: (1) modified blood, or (2) unmodified blood.

By modified blood is meant blood changed for the purpose of prolonging the time of coagulation so that it may be transfused before it becomes

never be followed by the immediate transfusion of whole blood, because *occasionally severe reactions occur.*

Whole Blood versus Plasma. In many conditions blood or plasma may be used interchangeably. For such conditions the use of plasma is preferable since it eliminates the necessity of blood matching. Another advantage is that it is simpler to store plasma than blood, and for a longer period. However, there are conditions in which one has an advantage over the other and there are others in which one or the other is contraindicated.

Let us remember that whenever there is loss of protein concentration of the blood, plasma is preferable to blood; whenever a great number of erythrocytes is lost, blood is preferable to plasma.

According to Strumia and McGraw the following are the indications for plasma and whole blood transfusion:

1. **Shock**

(a) With little or no hemorrhage—plasma should be given.

(b) With severe hemorrhage—plasma for immediate relief to be followed by whole blood.

2. **Burns** require plasma; whole blood is contraindicated because of hemoconcentration.

3. **Infections** require plasma to supply with specific and non-specific immune bodies. If, however, severe anemia is present, whole blood should be added.

4. **Blood dyscrasias.** In those with hemolytic tendencies, or with low prothrombin content, or with hemophilia, plasma should be administered. In those with hemorrhagic tendencies whole blood should be given.

5. **Anemia.** In various hypoplastic forms whole blood should be given; in chronic hypoproteinemia anemias plasma should be administered.

6. **Acute poisoning**—affecting the oxygen-carrying capacity of Hb. such as carbon monoxide poisoning—whole blood should be given.

UNMODIFIED BLOOD. By this is meant transfer of blood from the donor to the recipient without subtracting any constituents of the blood as is done in its defibrination and without adding any coagulants.

Theoretically, this type of blood transfusion is ideal; the only objection is that the blood coagulates before it is transferred into the vein of the recipient.

The methods employed for the transfer of unmodified blood can be classed into one of two groups: (1) The intermediary apparatus is lined with a substance which delays coagulation. To this group belongs the Kimpton-Brown tube,²⁸ the Percy tube, and like appliances. This method at one time enjoyed a great popularity in this country. Its good point is that it alters the blood very little, if at all. However, it has definite

disadvantages: the glass tube and the connection must be smoothly coated with paraffin; the procedure cannot be carried out without trained assistants; a vein has to be exposed surgically and ligated. (2) A measure, the success of which depends upon the rapid transfer of blood before coagulation takes place. The first who applied this method was Ziemssen in 1892; later it was revived by Lindemann²⁷ and others. In this method one person handles the syringe for the donor, the other—the syringe for the recipient and the third washes the syringes in saline solution and hands the syringes to the two operators. This requires a team of three workers who are not always available. However, this multiple syringe method was a forerunner of the syringe-valve methods in which instead of several syringes only one is used with the addition of a valve for changing the direction of flow from the donor to the recipient.

One of the earliest types of apparatus of this kind in this country was described by Unger,²⁸ in 1915. In the same year Bernheim²⁹ published a description of his apparatus which employed a syringe and a revolving plug valve. Since that time many modifications of the syringe and valve methods have been described. For this type of transfusion we use the apparatus introduced by Scannell^{30,31} and described below.

The technic of blood transfusion varies in accordance with the kind of blood used, namely modified or unmodified blood. Each of these two groups presupposes the employment of different instruments. But first of all the *proper selection of a donor* is of vital importance.

One must make absolutely sure that the donor is wholly free of any communicable disease and that his blood is compatible with that of the recipient. If non-compatible blood is transfused, the patient may die.

There are two methods of testing the compatibility of blood groups—the direct and the indirect.

The direct method is comparatively simple and therefore is the one suitable when the facilities afforded by hospitals or medical laboratories are not available. Under the usual aseptic precautions 2 to 3 cc. of blood is withdrawn from a vein of the recipient, collected in a test tube, and allowed to clot. The supernatant serum is removed with a pipette and one drop of it is deposited on a slide. Next a drop of blood is obtained from the donor after puncturing a finger or the lobe of the ear and transferred with the aid of a glass rod to the center of the drop on the slide. The slide then is gently rocked for 5 minutes and the drop is examined with the naked eye. If agglutination has taken place one sees small clusters but if agglutination is absent the drop appears slightly but uniformly tinted red.

If there is any doubt in the mind of the examiner, the final decision is made with the aid of the microscope.

Needless to say that whenever the cooperation of a trained pathologist can be secured, it should be obtained even for this simple procedure.

The indirect method necessitates the use of stock sera of blood groups

II and III. A slide is marked "II" at one corner and "III" at the other corner. One drop of stock serum II is placed under the spot marked "II," and another drop of stock serum III is placed under the correspondingly marked number. A drop of blood is obtained from the ear lobe of the recipient and transferred to the serum II by means of a glass rod. Another drop is now obtained and similarly added to the serum III. It is essential to make use of two separate glass rods, or else, if only one rod is used, to make sure that it has been carefully washed and cleaned after its use for the serum II before taking the drop of blood for serum III.

The slide then is rocked to facilitate mixing of the blood with the sera and examined with the naked eye. In the presence of agglutination the drop becomes clustered.

If agglutination does not take place after 5 minutes, the recipient's blood is compatible with either blood group II or III, as the result of the test shows.

TECHNIC OF TRANSFUSION OF MODIFIED BLOOD (SODIUM CITRATE) METHOD. The technic consists first of collecting blood from the donor, whose blood is compatible with that of the recipient and second of injecting it into the recipient. Each of these procedures is very simple and may be used with the aid of simplest appliances, such as a jar, rubber tube, needle and sodium citrate. However, on account of the importance of strict asepsis, of smoothness in drawing the blood, in mixing it with citrate and in preserving it sterile while it is injected into the recipient, it is much better to have sealed containers, which are not contaminated in ordinary handling and in which the necessary concentration of sodium citrate is present. The Baxter transfusion set is very efficacious. Each set is supplied with descriptive literature, which give in detail each step of collecting and injecting blood.

The Baxter unit provides a completely closed technic for collecting, citrating, transporting and storing whole blood. This unit consists of a hermetically sealed jar—Transfuso-vac and donor set (Fig. 20); the latter consists of a valve—vacutrol, tubing and needle. The transfuso-vac is of different sizes. It contains sufficient amount of anticoagulant which is mixed with the drawn blood in order to prevent quick coagulation.

The technic of collecting blood is as follows: After thoroughly shaking the transfuso-vac to coat the inside with the anticoagulant, the outer metal cap and the metal disc are removed as described in the section on venous infusion. Next, the donor set is unwrapped. The donor valve is now closed by turning the knurled knob clockwise. While holding this valve in the right hand the first or top rubber disc is removed, exposing the sterile surface of the second rubber disc (Figs. 21 and 22). The protecting vial from the valve needle is removed and, holding the transfuso-vac with the left hand, the needle of the donor valve is inserted per-

pendicularly through the center of the "X" mark on the stopper (Fig. 23). Then the protecting vial from the donor's needle is removed and the needle is inserted into the vein. Blood can be seen entering the transparent amber tubing. When the needle is in place the valve is opened by

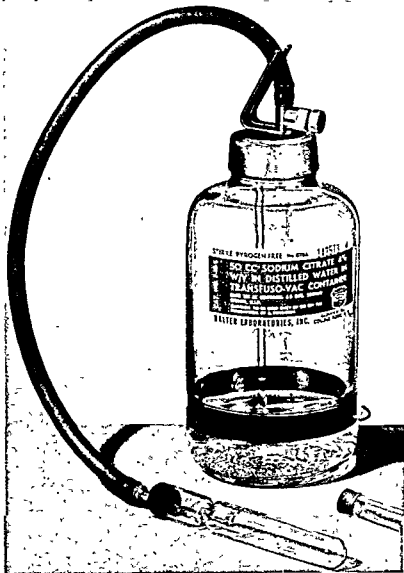


FIG. 20. Vacutrol donor set and transfuso-vac.

turning the adjusting screw anticlockwise. The blood will flow freely into the jar (Fig. 24). The rate of the flow will depend upon the degree in which the valve was opened. The average rate of flow should be between 75 to 125 cc. per minute. If the blood does not flow immediately, it is due to one of two causes: either the pressure of the tourniquet is

too high or the needle is not within the lumen of the vein. First, one has to manipulate on the presumption that pressure of the cuff of the sphygmomanometer is too high by reducing the pressure; if this does not

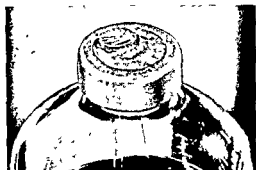


FIG. 21. Tamper proof seal of transfuso-vac.



FIG. 22. Exposed surface of transfuso-vac showing "X" under the remaining disc.

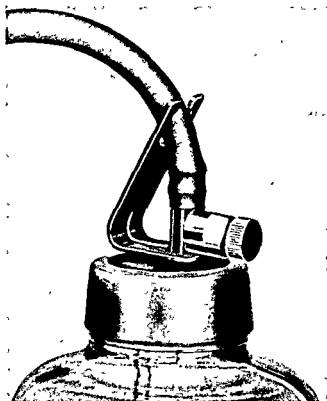


FIG. 23. Vacutrol valve inserted in transfuso-vac.

improve the flow of blood, the needle has to be reinserted. However, before starting to manipulate with the needle, the valve has to be closed; otherwise, if the needle is withdrawn and the valve is opened, the vacuum is lost.

When the blood is flowing into the transfuso-vac, the container is

agitated with a swirling motion to mix the blood and citrate. When the desired amount of blood is collected the valve is closed, the tourniquet is released, the needle from the donor's vein is withdrawn and the valve is removed from the stopper of the transfuso-vac (Fig. 25). After the valve is removed, the transfuso-vac is agitated for a minimum of three minutes

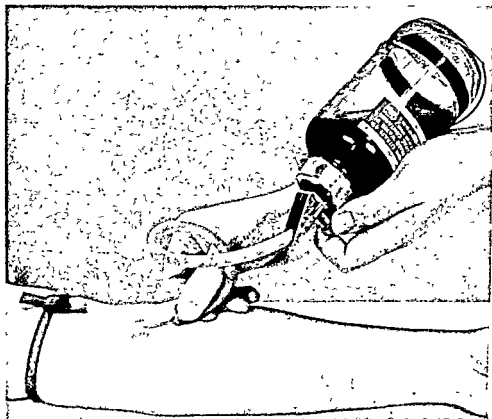


FIG 24. Collection of blood.

to insure an adequate mixture of the blood and anticoagulant. After the blood was collected the transfuso-donor set should be cleansed carefully. The instruction for this cleansing is usually sent by the laboratories in descriptive literature.

Administration of blood is the second act of blood transfusion, and is done in the following manner:

The remaining rubber disc on the transfuso-vac is removed. This exposes the rubber stopper, which is cleansed carefully with iodine and alcohol solutions. A sterile No. 18 gauge needle is inserted through the larger indentation on the stopper to dissipate the vacuum; then the needle is removed from the larger indentation and inserted through the smaller indentation, where it remains throughout the transfusion to

provide an air inlet. With the shut-off clamp on the tubing closed, the filter drip is inserted into the larger hole in the stopper. (The filter drip is a glass cylindrical tube interposed between the container and the tubing. It differs from vacodrip in this respect that it contains a stainless

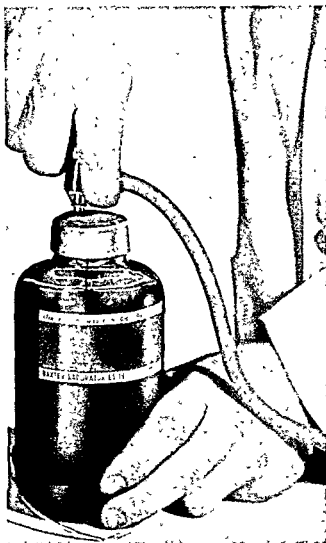


FIG.25 Removal of valve needle after blood collection.

steel screen acting as a filter and catching small particles, such as fibrin and any particles larger than 74 microns; in all other respects it has the same purpose).

The set is filled now with blood and the transfusion made exactly as for intravenous infusion (Fig. 26).

If it is desired to start and finish with normal salt or glucose solution, the same set may be used with a vacoliter filled with normal saline or 5 per cent dextrose in normal saline. The change is made from one to the

other by closing the tubing clamp tightly and inserting the filter drip into the other container.

THE ADMINISTRATION OF BLOOD, PLASMA OR SERUM COMBINED WITH NORMAL SALINE OR DEXTROSE SOLUTION. This set is seen in figure 27. It consists of a vacoliter, containing saline or glucose and transfuso-vac



FIG 26 Filling the filterdrip and infusion tubing preparatory to venipuncture.

containing blood or plasma. A "Y" tube connects each of these containers with a filter drip and tubing leading to the recipient. Three clamps on the infusion set are closed. The two containers are suspended and the infusion set is filled with saline (or dextrose solution) as follows:

The needle adapter pointing upward is held in the left hand; the clamp immediately below the vacoliter is opened and the tubing is raised high enough to invert the filter drip; the clamp underneath the filter drip is opened, so as to allow the solution to fill its chamber. The clamp beneath the filter drip is closed and the tubing is lowered enough so that the filter drip will acquire its right position (Fig. 27). The tubing

between the filter drip and lower clamp is squeezed, thus forcing the air into the filter drip. The lower clamp is opened again and the solution fills the remainder of the tubing

After the set is properly filled, the needle is attached and inserted in the vein. The blood (plasma or serum) may be given by simply opening the clamp on that side and shutting off the vacoliter.

In blood transfusion, often the rate of transfusion is slowed down even if the clamp regulating the flow has not been changed.

With this kind of tubing it is possible to close the clamp underneath the blood container and allow the solution from the vacoliter to flush the filter drip to restore the desired rate of infusion.

The most difficult part of the technic of transfusion is the introduction of the needle into the vein of the donor and particularly of the recipient, because larger needles are used for the recipient (No. 15 for recipient and No. 18 for the donor). The reason for using larger caliber needles for the recipient is that the needle stays in the vein of the donor only about eight minutes, whereas in the vein of the recipient it stays for about one to two hours. On the other hand, the vein of the recipient very often is collapsed and it would be more difficult to insert it in the recipient's vein even if the same gauge were used for both the donor and the recipient. The technic of inserting the needle into the vein is identical with that described in venous infusion. In some cases it is impossible to enter the vein of the recipient by the

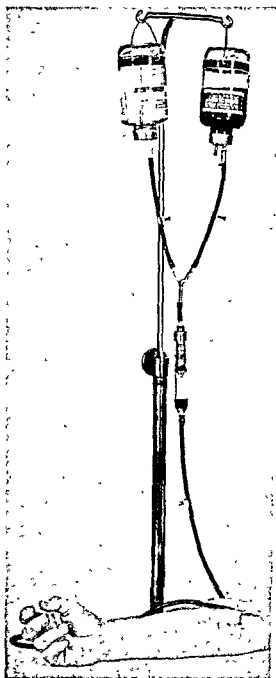


FIG. 27. Blood transfusion and saline infusion jars in the same set.

ordinary subcutaneous method and one is forced to use an open or surgical method. This is done in the following manner:

A cuff of the sphygmomanometer is placed around the upper third of the arm and is insufflated up to 60 to 70 mm. of mercury. (It should be

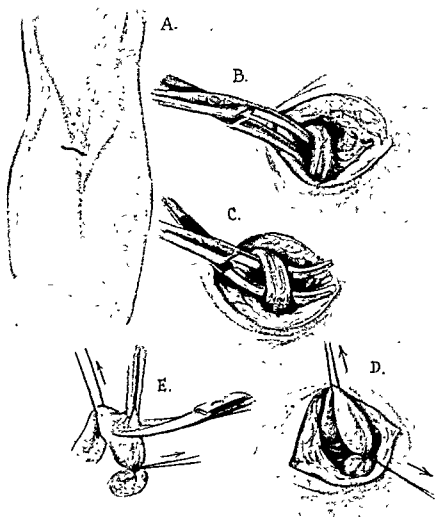


FIG. 28. Open method of inserting a cannula into a vein

slightly below the diastolic pressure.) The point is selected in the cubital fossa where the median basilic or median cephalic vein is situated. They are usually well seen after pressure of the sphygmomanometer is applied. This place is anesthetized by injecting 1 cc. of 1 per cent of novocain and well massaged. A transverse incision three-fourths

of an inch long and perpendicular to the course of the selected vein is made through the skin only (Fig. 28A). The upper and lower lips are retracted. The vein is now clearly seen. A small artery forceps with a curved pointed beak is placed at the side of the vein and the jaws are spread in a direction parallel to the course of the vein (Fig. 28B); this will break through the fat and connective tissue. The jaws of the beak are then closed and carried behind the vein; its tip breaks through the connective tissue on the other side of the vein (Fig. 28C). The jaws are opened again; a thread is caught and carried around the vessel; the same maneuver is repeated, thus having two threads around the vein

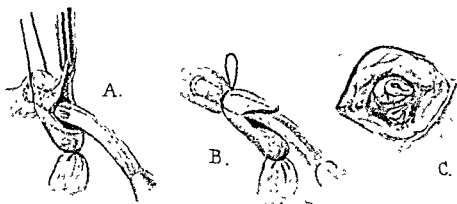


FIG. 29 Insertion of a cannula into a vein

at a distance of one-half inch from each other (Fig. 28D). The proximal thread is held with some tension, thereby shutting off the flow of the blood proximal to the ligature. The vein in the operative field becomes engorged. The thread at the distal portion of the vein is now tied while the thread on the proximal end is held taut. A snip with scissors cuts the anterior wall of the vein (Fig. 28E) and a needle or a cannula enters the lumen (Fig. 29A); the thread ties snugly around the groove of the cannula or needle by a bow knot (Fig. 29B) and the posterior bridge connecting the segments of the vein is now severed.

After the transfusion is done, the knot is dislodged, the cannula removed, the vein tied (Fig. 29C) and the skin closed by two interrupted sutures.

The transfusion of plasma is technically simpler than that of whole blood because the gauge of the needle is smaller and therefore easier to insert into the vein. There is no possibility of clogging the needle.

TRANSFUSION OF UNMODIFIED BLOOD BY SCANNELL APPARATUS (Fig. 30). The Scannell apparatus consists of a 20 cc. glass syringe and a triple

valve which is connected with the syringe and to three tubes: one leading to the donor, another to recipient and the third to the basin. The latter is filled with the normal salt solution and is used for washing the syringe, needles and cannulas. In case the operator breaks the original syringe, which comes with the apparatus, any Luer syringe can be used by attaching the valve adapter to it.

Preparation of the Apparatus. The syringe, tubing and needles wrapped in gauze are either boiled or sterilized in autoclave and kept ready for instant use. The size of rubber tubing is No. 18 French scale. To secure easy and free action of the glass syringe, the pistol and the inner side of the barrel should be lubricated with pure sterile vaseline. The apparatus

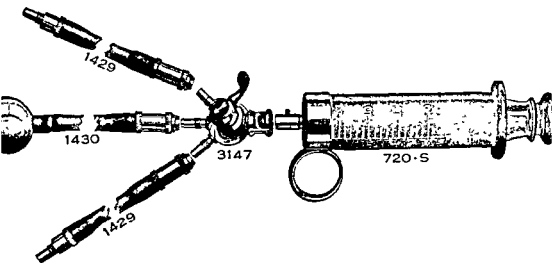


FIG. 30 Scannell Apparatus.

is assembled as shown in Fig. 30. The tube with the sinker is placed in the middle. A small basin of normal salt solution is provided. The air is expelled from the valve and tubes by filling them with the salt solution.

Preparation of the Donor and Recipient. The donor and recipient occupy operating tables of the same height. A small table or stand covered with a sterile towel is placed under their arms and between the tables or beds.

Actual Technic. A rubber tourniquet or sphygmomanometer band is placed around the arm of the donor and pressure brought up to 60 mm. The needle or cannula is inserted into the vein of the donor pointing toward the hand. Next, a side tube coming from the valve is connected with this needle and the tourniquet is loosened. Ten cc. of normal salt solution is drawn through the middle tube and injected into the donor in order to make sure that the point of the needle is resting wholly within the walls of the vein. A tourniquet is placed around the arm of the re-

recipient and a needle or a cannula is inserted into his vein, the needle pointing to the shoulder. Another side tube is connected with this needle. The tourniquet is removed and 10 cc. of normal salt is injected into the vein of the recipient to determine whether the needle or cannula are well within the lumen of the vein. Next, the tourniquet around the arm of the donor is again tightened to 60 mm. of mercury.

Transfusion. The transfusion should be started slowly. If the recipient shows any reaction, the transfusion is discontinued at once. The syringe is held in the left hand in such a way that the valve can be turned readily with the left thumb. The valve handle is turned until the arrow points to donor inlet; then with slight traction on the pistol the syringe will fill. (If it does not, either the needle is not in the vein or the tourniquet is too tight.) After the syringe is filled, the valve handle is turned in opposite direction until the arrow points to the recipient outlet and the blood from the syringe is next injected into the recipient's vein; the valve handle is pushed back toward the donor and these manipulations are repeated until the desired amount of blood has been transfused. After 150 cc. to 200 cc. of blood have been transfused, the valve handle is turned until the arrow points to the basin of salt solution and the syringe is washed out two or three times. Then transfusion is started again by pointing the valve handle to the donor's outlet and so on.

THE AMOUNT OF BLOOD TO BE TRANSFUSED DEPENDS ON SEVERAL FACTORS. One of the most important considerations is the indication for which blood is given. If it is administered for acute hemorrhage, the amount should be larger than if given for some chronic ailment and the rate of transfusion is much faster than that for a chronic ailment or as a preoperative measure.

In the average case 500 to 600 cc. is sufficient for acute hemorrhage; however, not infrequently 1,000 cc. to 1,500 cc. may be required in severe cases of shock due to hemorrhage.

If blood transfusion is done for raising the hemoglobin level in some chronic conditions, the amount of blood given should be able to raise the hemoglobin level to 45 per cent. If operation is contemplated, the hemoglobin level should be not lower than 60, preferably 80 per cent. For an average adult one pint of blood is able to raise the hemoglobin level by 10 per cent. A more exact estimation of the amount of blood needed for raising the hemoglobin level to a desired degree is given as a formula by Marriott and Keckwick:³²

$$\frac{\text{Per cent rise of hemoglobin required}}{100} \times \text{Patient's normal blood volume in cc.}$$

THE RATE of transfusion is subject to great variations: it should be neither too slow nor too rapid. It is given rapidly in case of shock—500

cc. in eight to ten minutes and slowly in case of chronic ailments—only 150 cc. per hour. On the other hand, in myocardial damage, the rate of flow of blood should be about 75 cc. per hour.

Complications

Though technically blood transfusion ranks a minor operative procedure, its complications and dangers connected with it may occasionally be those of a major operation. These complications may be due to reactions arising during or immediately after transfusion and which may, according to Kilduffe and DeBakey,³¹ be placed into the following classification:

I. *Reactions*

A. *Biologic*

1. *Hemolytic*

- a. Incompatibility
- b. Minor hemagglutinins
- c. Certain pathologic conditions
- d. Universal donor

2. *Proteolytic*

- a. Allergic
- b. Anaphylactic

3. *Incipient coagulative changes*

B. *Chemical*

1. Impure citrate or saline
2. Water not freshly distilled
3. Unclean apparatus

II. *Transmission of disease*

A. *Syphilis*

B. *Malaria*

C. *Miscellaneous diseases*

III. *Cardiovascular accidents*

A. *Cardiac failure*

B. *Embolism*

C. *Hemorrhage*

D. *Thrombosis*

REACTIONS. Reactions following blood transfusion were observed already since the seventeenth century. As mentioned in the historical introduction, Landsteiner, in 1900, discovered the presence in the blood of isoagglutinating and isoagglutinable substances which satisfactorily explained the occurrence of reactions and laid the foundation for its prevention. With improvement in technics the death rate due to transfusion steadily decreased and now there is about one reaction for 400 transfusions and about one death for 1,500 transfusions. Whereas the

mortality rate does not deviate greatly in the statistics of different investigators, the percentage of reactions varies greatly, being from one-fourth of 1 to 7 per cent. Evidently, the difference arises from different views of surgeons as to what constitutes a "reaction." In a severe *hemolytic reaction* the patient complains of a generalized tingling sensation, anxiety, fullness in the head, constricting feeling in the chest and pain in the back of the neck and in the lumbar region. The pulse becomes rapid, weak, the skin clammy; there is marked cyanosis, dyspnea, drop in blood pressure, nausea and vomiting. Immediately afterward, the patient has a chill followed by a rise of temperature. The reaction usually appears either at the time of transfusion or immediately after. It lasts about one hour. The fever may last twenty-four hours or less. The urine is red dark, due to hemoglobinuria. In a fatal case, this attack is followed by a few days of symptomatic improvement, followed later by the terminal stage of renal insufficiency. The patient sinks into a comatose state and death ensues within two weeks after the transfusion. The fatal rate in the hemolytic type is about 45 per cent.

The treatment for this type consists of stimulating the kidneys by intravenous hypertonic fluids, cardiac stimulants, by decapsulation of the kidney and plasma transfusion. However, the fact of the appalling mortality rate shows that there is nothing specific in all these measures.

Proteolytic reactions occur from time to time. They vary in intensity from mild to severe. Mild cases show urticaria, slight angioneurotic edema; moderately severe cases reveal difficulty in breathing, involuntary urination and defecation; severe cases resemble anaphylactic shock. The latter type (anaphylactic) is usually fatal; fortunately it is rare. The mild variety is relieved by an injection of 10 to 20 drops of 1:1000 of adrenalin.

Chemical reaction occurs from time to time. Some consider that sodium citrate may cause reactions; others absolve the sodium citrate but blame its impurity or the impurity of the saline solution, improperly distilled water or improperly cleaned parts of the transfusion sets, such as syringes, rubber tubes, and the like.

A reaction from chemical causes is probably the most frequent variety. It usually starts within one hour after transfusion, with a rapid rise of temperature up to 104° to 105°F. The temperature returns to normal within a few hours. The patient should be kept warm and morphine should be given. One may always try 5 to 10 minims of adrenalin (1:1000) on the assumption of an allergic state. The prognosis as to the outcome is good.

Reaction due to the Rh factor. This reaction occurs occasionally in women who have been pregnant some time before and in patients who have undergone repeated blood transfusions. The term Rh has been coined from the first two letters of Rhesus (*Macaca rhesus*) by K. Land-

steiner and A. S. Wiener who found that the serum of rabbits immunized with the blood of a *Macaca rhesus* will agglutinate not only the blood cells of monkeys but also of 85 per cent of humans, irrespective of the blood group of the latter. Accordingly, the Rh factor is an antigen contained in the red blood cells in the majority of human beings. Such individuals are said to be **Rh-positive** in contradistinction to persons who do not possess this antigen substance and who are therefore referred to as **Rh-negative**.

Rh-negative persons who repeatedly receive Rh-positive blood may develop agglutinins against it and sustain a severe reaction when given a transfusion of Rh-positive blood.

A like severe reaction also may occur in women who never before have been transfused but had been pregnant and whose blood as well as that of the fetus had opposite Rh factors.

These reactions have appeared even when in grouping and in cross matching the routine methods revealed no agglutination.

This discovery renders it imperative to determine the Rh factor of donors and recipients whenever one has a patient who on previous occasions had been given repeated blood transfusions or had been pregnant.

Transmission of Disease. Cases have been reported of transmission of communicable diseases through transfusion. Syphilis was the most frequent disease transmitted. Among others should be mentioned malaria and measles. Usually it is transmitted from the donor to the recipient, however, the opposite was the case on several occasions. In some of the cases of transmission of the syphilis this could have been prevented if the proper precautions had been taken; however, in some cases, the donors were in the preserologic stage and there were no clinical signs of disease. It should be remembered that in some cases of syphilis the Kahn or Wassermann tests are negative.

That the transmission of malaria may be facilitated is due to the fact that often the carriers of malaria not only do not know of it, but they even do not know that they have ever had malaria. In some cases it is not easy to demonstrate the presence of plasmodia in the blood.

Among other diseases transmitted are measles, relapsing fever, small-pox and influenza.

Cardiovascular Accidents. This is an extremely rare complication. In collected statistics of over 43,000 transfusions and quoted by Kilduffe and DeBakey³⁴ only six patients died from cardiac failure with pulmonary edema. Cases of embolism or thrombosis following blood transfusion are also extremely rare.

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Chapter II

Shock. Hemorrhage. Septicemia. Gas Gangrene

By GEORGE DE TARNOWSKY

(Revised by JULIUS L. SPIVACK)

SHOCK

Shock may be defined as a disturbance of functional equilibrium characterized clinically by reactions of depression which may be severe enough to prove fatal. Its two essential manifestations are subnormal temperature and low blood pressure. It is often associated with severe hemorrhages or precocious toxemias, increasing the difficulty of appreciating the relative importance of each etiologic factor. This is especially true in those cases in which both shock and hemorrhage are present in the same individual.

Theories of Surgical Shock

1. **Physical and physiological theory of Crile:** Shock is believed to be the result of exhaustion of the vital nerve centers.

2. **Chemical theory of Henderson:** Shock is caused by a loss of carbon dioxide plus the presence of acidosis. This is challenged by other investigators who deny that loss of CO_2 can produce shock and who have shown that the injection of acid initiates acidosis without producing shock.

3. **Organic theory:** Deficient adrenalin in the blood stream is advanced as the etiologic factor. It is now generally accepted that such deficiency is a result and not the cause of shock.

4. **Freeman** believes that shock is due to hyperactivity of the sympathico-adrenal system, thus accepting part of Henderson's theory.

5. **Swingle et al.** . . . , noticing that manifestations of shock and deficiency of the adrenal cortical hormone were similar, proposed the use of adrenal cortical extract in the treatment of shock. Other writers declare that there is no relation between the two and that furthermore the action of cortical extract is too slow to be of use in an emergency.

6. **Porter**, during the World war I, believed that fat embolism was the causative factor in shock. While it is true that in compound fractures of long bones—and occasionally in simple fractures—fat embolism is not uncommon and is the cause of shock in such cases, it does not account for shock in the absence of bone lesions.

7. The toxemic theory of Cannon is at present accepted by a great number of surgeons. Cannon states that it occurs as the result of the rupture, shredding, tearing or crushing of large amounts of tissue. Histamine from crushed muscles is liberated in the injured tissues, creating a vaso-motor paralysis. There ensues a dilatation of capillaries and a pooling of blood within them, leading to an abnormal permeability of the endothelial walls and an escape of plasma with concentration of corpuscles within the vessel walls. Acidosis is always present to some degree.

8. Edmund Andrews does not accept Cannon's theory and writes: "The general question of shock arising from toxins originating in injured tissues can, today, be answered with almost a categorical negative. Earlier reports of finding of histamine-like derivatives, acetylcholine, or various other toxic amines have, as a general thing, not been confirmed and to-day we feel fairly confident that the fallen blood pressure and collapse after mechanical or thermal injuries is rather definitely not due to absorption of poisons from the injured areas." Andrews is convinced that loss of circulating fluids is the most important factor in surgical shock, but he fails to give any explanation regarding the cause of this leakage of blood plasma into the loose cellular tissues.

In the mechanics of circulatory failure three important factors are to be considered:

1. **PERIPHERAL.** A capillary and arterial dilatation will, of course, bring about a fall in peripheral resistance and hence a drop in pressure. This factor is largely dependent in most cases on the tone of the autonomic and sympathetic nervous systems, and may occur as a reflex phenomenon.

2. **CARDIAC FAILURE.** If the engine stops or falters, quite naturally the pressure will correspondingly drop. Strange to say, however, in most of the conditions with which the surgeon has to deal, the heart itself is not at fault.

3. **LOSS OF CIRCULATING FLUID.** Blood pressure falls from external hemorrhage is an obvious matter. However, blood pressure falls from multiple internal capillary hemorrhages following trauma have only recently been recognized. Furthermore, it is quite possible for plasma to leak into the tissues under certain stimuli and cause a corresponding loss in circulating fluids (Andrews).

Psychic shock was a not uncommon occurrence during the war and should always be considered in the differential diagnosis. It is essentially a defense reaction, a means of avoiding or refusing to meet a sudden situation, sorrowful, disagreeable or dangerous. It differs from traumatic shock in that, while there is a profound fall in blood pressure, there is no capillary dilatation and a marked bradycardia and not a tachycardia is present. There is a transitory cardiac shock with cerebral

anemia and hysterical outbursts. Placing the patient in a straight-back chair and lowering it so that the legs and feet are elevated while the head and trunk are lowered horizontally is the only logical treatment. Stimulants are unnecessary; those belonging to the vaso-motor group may even be detrimental.

The existence or non-existence of pure neurogenic shock has been debated, pro and con, for many years. Whether prolonged stimulation of sensory nerves can bring about a fall in blood pressure, has been answered affirmatively and negatively. While the existence of neurogenic shock cannot be entirely denied, its importance is, in all probability, slight.

During operations under general anaesthesia a rather sudden collapse with marked lowering of blood pressure may occur. When not caused by obstruction of the upper air passages, it denotes an inhibition of oxidation, producing tissue anoxemia. High oxygen concentrations may help in some cases. Likewise, if the anesthetized patient rebreathes from the anesthetic bag gases deficient in carbon dioxide, hyperventilation of the lungs or acapnia (Henderson), will soon appear. Henderson showed that the presence of CO_2 in the blood was the normal stimulant of respiration and not the lack of oxygen. A deficiency in CO_2 will bring about a cessation or diminution of respiration shortly followed by collapse. The administration of concentrated CO_2 will restore normal respiration.

In extensive, especially comminuted, fractures of the long bones, multiple tiny fat droplets from the bone marrow may accumulate in the pulmonary capillaries and, if the quantity be sufficient, bring about some degree of pulmonary blockage leading to rapid shock and collapse. The treatment of this type of shock is entirely empirical. Herrmann reported success through using an intravenous injection of seven parts of a 25 per cent dextrose solution and three parts of 95 per cent alcohol. He advises using 5 cc. per kilo of body weight which he estimated to be about 25 per cent of the lethal dose.

Etiology

The clinical factors producing the symptom complex of shock may be conveniently divided into exciting causes on the one hand and contributing or accentuating factors. Trauma, whether accidental or surgical, hemorrhage, precocious septicemia and nervous shock are all exciting causes. Nervous shock may be further subdivided into anxiety neuroses, hysteria and, rarely, malingering. Of the contributing and accentuating factors pain, cold, hunger, thirst, heat, fatigue, loss of sleep, injury to important viscera, multiplicity of injuries and psychic influences must all be evaluated, as any of them may necessitate a modification in our treatment. The importance of recognizing these secondary factors was most dramatically demonstrated during the World war I. In the spring

and summer months of 1918, when the weather was comfortable and our soldiers were fresh from training camps, shock only occurred among the severely wounded; when the rainy season opened and our troops were fighting in the Argonne sector with transportation bogged up, inadequate food and shelter, loss of sleep, cold and hunger became almost *major factors in the production of shock regardless of the type of injury received*. The importance of preventing or minimizing these same factors in civil life will be taken up in the outline on treatment.

Air embolism is a constant possibility in operations or traumas around the neck, head or axilla where air may be sucked through a torn vein into the circulatory system by respiratory motions. Death is probably caused by a sudden filling of the right auricle and ventricle with froth. Auscultation of the heart will reveal a characteristic "shushing" sound which is pathognomonic. The only logical treatment—often ineffective—consists in aspirating the right heart with a large hypodermic needle and giving heavy doses of cardiac stimulants.

In biliary surgery, if the surgeon rotates the liver for a better exposure of the biliary tract, a complete portal obstruction may result with a corresponding, often fatal, dilatation of the abdominal veins. Should the anesthetist report a sudden drop in blood pressure, the liver should immediately be replaced in its normal position.

Symptomatology

The essential manifestations of shock are: Cold sweat, cold extremities, clammy skin, white, pale grayish or slightly cyanotic appearance; a great thirst and often vomiting. The temperature is subnormal, the pulse of low tension, variable rapidity and feebleness, with weakened heart sounds; partial or complete muscle flaccidity is common, the pupils are dilated and there is lessened or total loss of sensibility. The respiration is usually rapid but may be either depressed or shallow. Subnormal temperature and lowered blood pressure are the cardinal findings. Patients are first restless and very anxious but lapse gradually into a listless or apathetic state. In either primary or secondary shock oligemia is present to a certain degree. By this term is meant a condition in which the volume of circulating blood is diminished. When this is so far reduced that the various compensating mechanisms are unable to maintain a normal effective venous pressure in the right auricle, the effective pressure in the left auricle also falls. In consequence the output of the left heart diminishes and the arterial pressure tends to lower. Such a tendency prevails although countered by a reflex peripheral constriction. Hence both systolic and diastolic pressures are low, the pulse pressure is small, and the product of heart rate and pulse pressure decreases (Wiggers). A persistent diastolic arterial hypotension of less than 60 mm., independent of hemorrhage or toxemia, is an index of the gravity

of the shock and the principal indication for its treatment. The effect of the treatment can only be correctly estimated by repeated applications of the sphygmomanometer. Repeated blood counts may be necessary in certain cases where the possibility of an internal hemorrhage cannot be ruled out. In hemorrhage there is loss of whole blood with a consequent reduction in the number of red blood corpuscles and hemoglobin because the vascular bed has absorbed fluid from the surrounding tissues to increase the blood volume. This only occurs when the amount of blood loss cannot be compensated by increased heart action and peripheral vaso-constriction. In shock on the other hand, there is loss of circulating blood volume and the blood has been pooled in the capillary bed because of capillary paralysis. The arterioles are constricted, and although there has been no loss of whole blood out of the vascular bed, the stagnation of blood in the capillaries reduces the circulating blood volume. The loss in shock is *serum and not whole blood*, therefore the red blood corpuscles and hemoglobin are either normal or actually increased.

Moderate abdominal-wall traumas are associated with varying degrees of shock, regardless of the presence or absence of visceral lesions. The well-known solar plexus blow of pugilism is a striking example of sympathetic shock. Initial shock, however, cannot be used as a criterion in determining the amount of damage done. Examination of the abdomen will often reveal the nature of external trauma (bruise of an automobile or truck wheel, contusion of the skin, beginning hematoma, early manifestations of supra-pubic extravasation of urine in extraperitoneal rupture of the bladder or bleeding from the penis or female urethra in intra-peritoneal bladder rupture, subcutaneous emphysema of the thorax and abdomen in fractures of the ribs with injury to pleura and lung, etc.). One should ascertain about when the patient had his last meal, when he last defecated or urinated. The answer to these questions often has an important bearing on both diagnosis and prognosis. Should the initial shock tend to be prolonged, with symptoms of hemorrhage (fullness in the flanks, shifting in character, increase in pallor, air-hunger, etc.), a tear of a mesenteric vessel or rupture of a solid viscus should at once be surmised. Pain is a very variable factor. Simple contusions of the abdominal wall are frequently more painful than visceral lesions—until peritonitis sets in, when the condition of the patient undergoes a rapid change for the worse. Early emesis is not important. Pernicious emesis, blood-tinged or not, suggest a severe visceral lesion. Persistent hematuria strongly suggests a ruptured kidney. Hematuria followed by anuria is typical of a recent intraperitoneal or of a three-to-four day old extraperitoneal rupture of the bladder. Progressive abdominal distention is strongly indicative of rupture of a hollow viscus. The value of a fluoroscopic determination of free gas beneath the diaphragm or scattered beneath the various peritoneal pouches was pointed out by

Vaughan and Rudnick and is a test which should be promptly used in all doubtful cases. In a typical case of intra-abdominal trauma, following the initial shock, the patient has an anxious facies and becomes restless with pulse of increasing rate and decreasing tension. The extremities become cold and clammy. The hepatic dullness disappears as abdominal distention and rectus rigidity increase. Respirations are rapid, shallow and painful. Unfortunately it is only rarely that one finds all the above clinical symptoms in a given case. The wise plan is to proceed slowly. A chart of the pulse rate, volume and pressure recorded every half-hour, and blood counts, will soon help to confirm a diagnosis of hemorrhage, even in the absence of shifting dullness in the flanks. A diagnosis of gastric or intestinal perforation is often difficult to make prior to the onset of peritonitis. The x-ray is invaluable in such cases. The severity and persistence of abdominal wall pain and distention are the two most valuable signs. Rigidity may, during the first few hours remain unilateral or localized, but it soon tends to become bilateral and generalized. Lejars taught that in simple shock abdominal distention was instantly universal, whereas in perforation the distention was first local or segmental and only later general.

Treatment

Transfusion of plasma or of whole blood is a life saving procedure in many instances, irrespectively, whether it is a sequela of hemorrhage or not. In case if it is due to loss of blood, blood transfusion is preferable to plasma transfusion; if there is no hemorrhage, plasma transfusion is preferable to blood transfusion as in shock already hemoconcentration is present. The amount of plasma or blood given varies depending on severity of the case. In average 500 cc. of plasma is sufficient. However, cases are reported in which up to 4,000 cc. of plasma was given until the patient was brought out of shock. In addition to this, other steps has to be taken. The patient's head should be lowered, except in head or thoracic injuries. Heat should be applied to the entire body by means of hot blankets, hot water bottles or a cradle with electric bulbs attached. A temperature of 104 to 106 degrees is thus quickly obtained and should be maintained until a clinical reaction is manifest. Hot liquids by mouth or rectum should be given freely. Minimizing of additional traumatism is very important; all fractures and extensive lacerations should be immobilized by suitable dressings and splintings at the time the patient is being given first-aid. Bandaging of the extremities, if properly done, is useful; proximal constriction from improper bandaging is worse than useless. The proper method is first to elevate the limbs in order to restore blood to the central circulation and then, with the limbs still elevated, to apply an elastic roller bandage beginning at the toes and fingers and

running up to the trunk, so that the blood is kept out of the extremities. This method is equally beneficial in the treatment of hemorrhage. If, as the patient recovers from his initial shock, he complains of severe pain, $\frac{1}{4}$ grain of morphine should be given hypodermically and repeated as needed. Absolute physical and psychic rest must be enforced. Normal saline or Ringer's solutions should not be used in shock because the fluid merely escapes into the loose cellular tissues. A 6 per cent gum-acacia solution of known purity is preferable because its molecules are too large to escape through the vessel walls.

Check any serious, visible, external bleeding with forceps, ligature or suture. Tourniquets should be avoided whenever possible but, if used, should be loosened every half hour for five minutes until proper surgical instruments are available. Gangrene of an entire extremity has resulted after three hours of continuous tourniquet compression.

Whether shock should be considered an indication or contra-indication to operation depends on the severity of the shock, presence or absence of hemorrhage and the type of operation contemplated. If hemorrhage can be ruled out or, if external, has been controlled, and the patient is cold and pulseless, shock should be treated first. This also holds true when the operation is a long and complex one such as a laparotomy with multiple visceral lesions. An extensive laceration of a limb, necessitating amputation, constitutes an indication for immediate operation with combined shock treatment. Fractures, either simple or compound, should not be permanently reduced until shock has been controlled. Thorough débridement of lacerated tissues and removal of all completely detached spicules of bone should be undertaken with the shock treatment. Ethylene or cyclopropane should be the anesthetics of choice; ether is less desirable in shock. According to Bowlby and others, spinal anesthesia is dangerous while shock is present.

HEMORRHAGE

Urgent surgery only concerns itself with hemorrhage of sufficient volume to endanger life.

Classification

1. Closed wounds
 - Contusions
 - Crushing injuries
2. Open wounds
 - Punctured
 - Lacerated
 - Incised
 - Combined arterio-venous

3. According to time

Primary

Intermediate (reactionary)

Secondary (latent)

4. According to kind

Arterial

Venous

Capillary

1. **CLOSED WOUNDS.** Some projectiles (bullets, metal fragments, etc.) or heavy weights such as trucks, wheels, cases, rocks, etc., may confuse or crush an artery or vein without producing any loss of blood through the vessel wall. Any lesion of the intima is likely to be followed by *thrombosis of the injured vessel*; unless *collateral circulation is totally inadequate*, many of these thrombosed vessels are unrecognized unless clinically revealed by embolic complications, aneurysms or secondary hemorrhage. Several days may elapse between the injury and appearance of vascular dysfunction. Whenever a wound has been received in a vascular portion of the body, the mere fact that there is no external bleeding will not prove the absence of a vascular lesion.

2. **OPEN WOUNDS.** Regardless of the type of open wound inflicted on a blood vessel, the clinical manifestation of hemorrhage will only vary in quantity, depending on the degree of gaping of the wound, size of the artery or vein, presence or absence of oxygen and, most important of all, on the amount of resistance to the outpouring of blood offered by the surrounding tissues. Hemophilic, icteric and diabetic patients present added problems.

The cardiodynamic picture following the rupture of an artery is dominated in the initial stages by a decrease in resistance and, in later stages, by the effect of reduced venous return. Initially, diastolic pressure decreases more than the systolic, the pulse pressure is greater and the *peripheral pulse acquires a collapsing characteristic*. The following mechanisms tend, to some degree, to compensate for the sudden loss of blood: (1) Diversion of blood from capillary pools, (2) contraction of the spleen and (3) dilution of blood by absorption of lymph. The tendency for arterial pressure to fall rapidly as a result of hemorrhage is partly counteracted by: (1) an increase in depth and rate of respiration which tends to favor the return of blood to the right heart, (2) an inhibition of the *vagus center causing an increased heart rate* and (3) a stimulation of the *vaso-motor center causing a constriction of peripheral blood-vessels* (Wiggers).

Every hemorrhage, except one coming from a very large vessel has a natural tendency to be checked long before the loss of blood becomes dangerous. The duration of bleeding depends not so much upon the

size of the vessel wounded as on the facility with which an efficient clot forms over the opening and on the type of wound, open wounds exposed to the air greatly facilitating the phenomenon of blood-clotting. In closed wounds the size of the hematoma will, to a large extent, depend on the anatomic barriers tending to circumscribe the effusion. Closed wounds involving the dome of the liver are rarely fatal because the bleeding is arrested by pressure of the diaphragm above and falciform ligament laterally. The same is true of lung parenchyma, muscle, fascial planes or subcutaneous connective tissue. It thus becomes easy to understand why retroperitoneal, uterine, splenic or mesenteric wounds so often demand immediate radical surgical intervention.

In closed abdominal wounds the organs most often ruptured are the liver, spleen, kidney and small intestine; the mesentery is not infrequently torn. The colon, rectum and bladder are less often injured. Stab or bullet wounds are frequently multiple, hence more confusing.

Diagnosis

The cardinal symptoms of hemorrhage are a rapid pulse, easily compressible, a low blood pressure and obvious anemia. Excessive thirst and air-hunger are common. If the bleeding is severe and intra-abdominal, fluid may be detected in the flanks and shifting dullness may be demonstrated by turning the patient from side to side and percussing the dependent portions of the abdomen. They may state that they feel "something hot running inside." We have frequently noted this particular symptom which is hard to explain physiologically, for the temperature of blood, urine or gastric content is that of the body, and yet some patients are conscious of something hot running through the abdominal cavity and free blood is found at operation. A description of the accident, when obtainable, is of great value in determining the probable presence or absence of hemorrhage. Hematuria suggests a ruptured kidney; the passing of fresh bloody urine, spontaneously or per catheter, is almost pathognomonic of a ruptured bladder. Bloody feces or a muco-blood discharge make one suspect a laceration of some portion of the intestinal track. Frothy hemoptysis suggests crushing injury of the thorax. If the site of the abdominal trauma is over the right or left upper abdominal quadrant, either the liver or spleen may be torn. Trauma over the epigastrium may have involved the stomach, transverse colon, duodenum or pancreas, whereas severe contusion in the vicinity of the umbilicus may be accompanied by injury to the small intestine or some segment of its mesentery.

Prognosis

The prognosis should always be guarded and usually kept in abeyance for at least twenty-four hours after surgical intervention. It not only

depends on early intervention when indicated, but also on the number and severity of the visceral lesions, ease with which bleeding can be checked and the possibility of complications such as peritonitis, visceral necrosis, etc.

Treatment

Basic indications for immediate operation are clinical symptoms pointing to progressive intra-abdominal hemorrhage or hollow visceral penetration. As pointed out in the treatment of shock, often repeated blood counts are invaluable. Injuries involving the main trunks in the neck, thorax or abdomen, and those of the main vessels of the liver, spleen or kidneys are usually fatal before surgical help can be given. When the injury has been brought about by a lateral blow, fall or crushing, a rupture of the liver, spleen or either kidney must be considered. A midline or para-umbilical blow is more apt to produce intestinal trauma (rupture, tearing of mesenteric vessels). Rupture of a hollow viscus demands immediate intervention, even though signs of hemorrhage are absent. With a suspected rupture of a solid viscus, but without alarming symptoms of hemorrhage, an expectant attitude should be adopted. The following case well illustrates this point:

A-3429, Ravenswood Hospital 12/29/1938. Referred by Dr. J. S. Schriver.

A four year old boy was caught between the bumpers of two automobiles and his lower thorax squeezed before the cars could stop. When admitted in the hospital he was in mild shock. T.99.6, P.110.R.40. His blood count was: Reds 4,500,000, Whites 27,100, Hemoglobin 77 per cent Sahli. An x-ray of the thorax was negative as to fractured ribs or elevation of the diaphragm. A few hours after admission he suddenly went into profound shock with marked air-hunger. Oxygen inhalations were immediately started and consultation asked for regarding the advisability of giving the child a blood transfusion. Percussion of the lower thorax on the right side revealed some impairment of dullness with no shifting quality. Paracentesis revealed free blood between the dome of the liver and the diaphragm. A blood transfusion would have merely encouraged a secondary hemorrhage and it was decided to adopt a "watchful expectancy" attitude. The boy convalesced rapidly and left the hospital on the 9th day. No further aspirations were made and rapid absorption of the hematoma occurred.

Most of these patients recover unoperated. If the hemorrhage has been severe and progressive, the best working rule is to operate if the patient is seen very early, but to watch and observe for a few hours if the patient is seen six or eight hours after injury. If then the anemia is not apparently progressive one should not operate, for the hemorrhage has to all intents ceased and laparotomy will cause a renewal of the hemorrhage with a probable fatal result. On the other hand, if there are signs of progressive bleeding, the abdomen should be opened at once. A good working principle is to operate if reasonable doubt of a hollow visceral lesion exists, for the mortality of such operation is exceedingly

low when no visceral penetration is found. Such a borderline case is that of:

68612, Ravenswood Hospital 3/14/1933.

D. L. Male, 26 years old, was knocked down by an automobile and brought to the hospital complaining of marked pain over his abdomen and a feeling as if "something hot had been poured into his abdomen." The skin over the left lower abdominal quadrant was contused; no fractures were discovered. Urinalysis was negative. T.98., P.120., R.30. Blood pressure 120 over 80. There was marked abdominal rigidity with, apparently, some dullness in the left flank. The blood count was: Reds 4,700,000, Whites 11,600, Hemoglobin 85 per cent Sahli. The pre-operative impression was that we were possibly dealing with a mesenteric tear with hemorrhage. What was found was a descending colon and sigmoid edematous and covered with petechial hemorrhages. No large vessel had been torn and there was no hollow viscus ruptured. The abdomen was closed without drainage and the patient made an uneventful recovery. There was a reasonable doubt in this case and it is always safer to explore and find nothing than to procrastinate until it is too late to save a life.

Two general rules may be followed to aid the surgeon in his choice of an incision: (1) Plan the incision to meet the visceral injury suspected; (2) Avoid, if possible, the entrance wound site—if any be present—and so diminish the liability to wound infection. Always make the operative wound sufficiently ample to insure an unhampered exploration. A 5 to 6 inch incision avoids evisceration of intestines during repair of same. For all middle and lower abdominal operations the paramedian incision gives the best results. For injuries to the liver or spleen, without trauma to hollow viscera, the trans-thoracic, extra-pleural incision facilitates exposure and treatment of these two solid organs. The latter technique is based on the anatomic fact that a thoracic incision beginning at the eighth costal cartilage in the para-sternal line and running obliquely downwards, outwards and backwards to the tenth rib in the mid-axillary line and twelfth rib in the scapular line, absolutely avoids the pleural sinus and enables the surgeon to incise the diaphragm on either the right or left side and expose liver or spleen. A lumbar incision is only indicated in kidney lesions or retroperitoneal hemorrhages. In the presence of free intra-abdominal bleeding a suction apparatus should be used. Many valuable minutes will be saved if the surgeon has a definite routine for his examination of the abdominal contents. The writer's preference is to begin at the right upper abdominal quadrant, palpating the right kidney, then passing a hand over the dome of the liver, its anterior and inferior surfaces. A finger is next introduced through the foramen of Winslow in order to palpate the head of the pancreas and first two portions of duodenum. The stomach and gastrocolic omentum are then inspected for rupture or laceration. The spleen and left kidney come next. By lifting up the great omentum and transverse colon and passing an index finger from right to left under the colon, the first loop

of small intestine encountered is at the ligament of Treitz. From that point on successive loops of small intestine are lifted up, carefully examined and rapidly replaced in the abdomen by an assistant. Having arrived at the ileo-cecal valve the entire colon is inspected to its pelvic end. Finally the bladder and female genitalia in women are examined for possible trauma. Any area where trauma is encountered is temporarily tamponed or covered with a laparotomy sponge. If a hollow viscus is torn the involved loop is doubly clamped off with intestinal clamps and the perforation closed. Blood vessels, if torn, are clamped. Having thus completed a thorough examination, nothing can be missed and the surgeon can now proceed with the permanent care of the lesion or lesions encountered. This complete preliminary survey is of particular importance in cases of gunshot or stab wounds where lesions are so apt to be multiple.

Wounds of the liver are tamponed, the end of the tampon brought out through a right lumbar stab wound and progressively withdrawn after five to seven days. Suture of the liver is rarely possible; it usually increases the bleeding.

With the exception of small tears which merely require tamponing, a laceration extending to the hilus of the spleen usually involves one or more of the splenic vessels and necessitates splenectomy. Care must be taken not to injure the tail of the pancreas or greater curvature of the stomach while ligating the splenic pedicle. In poor surgical risks the pedicle can be double clamped with eight inch forceps which are then covered with vaseline-coated gauze and left in situ for three to five days.

Tears involving the mesentery at right angles to the long axis of the intestinal track seldom require more than double ligation of the arterial or venous branch and closure of the rent. Where there is complete detachment of a segment of bowel from its mesentery and the proximal portion of the mesentery has had its main artery torn, the prognosis as to viability of the segment is bad. Collateral circulation is notoriously poor and the surgeon must decide between an immediate resection and end-to-end anastomosis in good risks and exteriorization of the involved loop in bad risks. In the latter case tubal drainage proximal to the exteriorized loop must be provided. In any case the torn vessel must be double ligated, even if the bleeding has ceased.

Unless the renal artery has been severed, hemorrhage from either kidney will rarely warrant a primary nephrectomy. Tamponing a kidney laceration and bringing out one end of the tampon through a lumbar stab wound will usually suffice as far as immediate treatment is concerned. Hemorrhage from a ruptured bladder is never serious per se, but immediate repair will prevent a deep cellulitis or peritonitis, depending on whether the tear is extra or intra-peritoneal.

HEMORRHAGE FROM THE STOMACH. As a rule bleeding from injury to

the celiac axis is rapidly fatal. A wound of one of the main gastric arteries may be followed by necrosis of a portion of stomach wall. After checking the bleeding by double ligature, apply hot packs to the injured wall for five or ten minutes; if the circulation is reestablished, as evidenced by a return to the normal pink-red of the stomach wall, it is safe to close the abdomen without drainage. If the segmental circulation appears jeopardized, leave a cigarette drain for three or four days and watch the clinical course of the case. At the first sign of local peritonitis, reopen the incision and treat the gangrenous section as indicated by actual conditions found. Wedge-shaped excision, partial gastrectomy or a gastro-enterostomy after removal of the gangrenous area and closure of the gap may have to be performed; no rules can be laid down. One is rarely justified in performing a radical operation on a wounded stomach as a primary measure; the immediate mortality is too great.

Bleeding from a wound of the pancreas is not, per se, serious but the escape of pancreatic ferments in the peritoneal cavity represents one of the hardest problems in surgery. The sheltered position of this organ makes it almost impossible to be wounded without adjacent injury to the stomach, transverse colon or spleen. An early pre-operative diagnosis of pancreatic lesion is impossible. When discovered, one may—rarely—attempt to suture the rent. Usually the surgeon will be limited to tamponing of the tear, large tubal drainage and protection of the abdominal wall by means of zinc-oxide ointment generously spread over the entire abdomen. The mortality is high: of the cases which survive a small percentage will develop a pancreatic cyst.

Blood transfusion, 5–10 per cent glucose solutions, heat to the body, hot drinks when tolerated and complete physical and psychic rest are all valuable adjuvants.

SEPTICEMIA

The terms **Pyemia**, **Sapremia**, **Septicemia** and **Bacteremia** are somewhat loosely used to denote clinical departures from the normal. From the pathologist's viewpoint, the products of bacterial action may be absorbed into the blood, giving rise to sapremia, or the bacteria themselves may enter the blood stream, a condition of septicemia.

Sapremia is a condition of intoxication due to the action of saprophytic bacteria. These are usually unable to invade the living tissue but flourish in dead tissue such as occurs in a wound with extensive laceration of the soft parts or in the remains of a placenta retained in the uterus. Owing to the great proteolytic power of the saprophytes, the proteins are decomposed into toxic products which, when absorbed, produce symptoms of intoxication. When the toxin factory is destroyed, as by scraping out the uterus or "débridement" of a wound, the symptoms rapidly subside.

Septicemia is a term familiar to everyone, and yet, when we analyze it, we find it difficult to define. Strictly speaking, any condition in which microorganisms circulate in the blood is a septicemia. In a clinical sense it is certainly not true. We must distinguish between a bacteremia and a septicemia. Bacteria probably enter the blood in every infection, but they are speedily destroyed and are therefore not found in blood culture. Moreover it is not only in infections that bacteria circulate in the blood stream. When a boy receives a blow on the shin and, as a result, develops acute osteomyelitis of the tibia, the infection is admittedly a hematogenous one and the circulating blood must therefore have contained bacteria at the time of the injury. Such a person is suffering from a bacteremia but not a septicemia. The difficulty is best solved by using septicemia as a clinical and not a pathological term; "it indicates a condition characterized not only by the presence of bacteria in the blood, but also by the development of certain clinical manifestations (pyrexia, petechial hemorrhages, etc.) suggesting the presence of these bacteria. Into every case of septicemia there should enter the conception of some local focus of infection, often undiscovered, from which, as a result of a breaking down of the protective barrier between bacteria and host, organisms enter and continue to enter the blood stream. When the source of supply is removed, the bacteria soon disappear from the blood. Prognosis, to a certain extent, may be based on the number of circulating bacteria as shown by plating blood cultures" (Boyd). The Kocher-Ravel classification which, unfortunately, is not currently used, is the most precise. It recognizes three clinical types of general intoxication:

1. A general intoxication by bacterial products without the presence of bacteria in the circulation, a "toxinemia" such as is found in tetanus and gas gangrene.
2. A general intoxication in which bacteria gain access to the general circulation, a "bacteremia." Where no metastatic foci develop, the term septicemia applies; should such foci develop, as in a case of puerperal streptococci infection, the term pyemia should be used.
3. A general intoxication provoked by toxins elaborated by bacteria which do not enter the circulation, associated with a general infection caused by bacteria which do enter the circulation. Such a mixed infection, typified by a combined diphtheritic and streptococcic invasion should be termed a "toxibacteremia."

Etiology

Predisposing factors which tend to reduce the protective forces of nature in an individual, such as excessive fatigue, anemias, diabetes, nephritis, etc., play an important rôle in the production of septicemia. Prolonged operations, rough handling of tissues, especially visceral peritoneum, uncalled-for and usually unnecessary exposure of viscera dur-

ing an operation, all tend to lower resistance. It is not improbable that, taking advantage of a lessened degree of immunity, saprophytic bacteria cease ingesting dead cells and, attacking living body cells, become pathogenic in the clinical sense, returning to their former saprophytic existence when the balance of power is restored. The writer believes that, in a majority of instances at least, these parasites are not suddenly introduced from without but that, taking advantage of either a generalized decrease of natural immunity or of a "*locus minoris resistentiae*," they wander from their normal habitat in the body, suddenly become activated and produce abnormal quantities of toxin.

While staphylococci, pneumococci and colon bacilli may be found in these generalized infections, the streptococcus is responsible for the vast majority of them. In 210 cases analyzed by Lenhartz, 160 cases of septicemia were streptococcic, 22 staphylococcic, 20 due to the pneumococcus and only 8 revealed colon bacilli as the active agent.

One must also bear in mind that an apparently simple pyemia may suddenly change into a septicemia and, conversely, an apparently fulminating septicemia may subside and become a pyemia with multiple metastatic abscesses. Pyemia and septicemia cannot be considered as two separate entities; they merely represent varying reactions of an organism more or less resistant to a microbe more or less virulent. Were it clinically possible to correctly evaluate the power of resistance of a given individual and the degree of virulence of the pathogenic invader of that same individual, a fairly accurate prognosis might be possible.

Symptoms

The onset of septicemia may be so rapid that, even when the primary focus of infection is discoverable and removable through extirpation, drainage or relief of tension, little or no improvement may follow. This is particularly true of gas gangrene cases. The outcome will almost entirely depend on the rapidity with which the individual can marshal his own natural defenses. It is in such cases that therapeutic attempts to lower fever are to be deplored. From a clinical viewpoint there can be no question but that fever is our best index of the virility of reaction on the part of the patient against bacterial infection. As Da Costa tersely states it: "Fever is only a symptom and the elevated temperature of an infection is evidence that the body is fighting the infection. An acute infection with a low or subnormal temperature is a far graver condition than an acute infection with a high temperature. The low temperature shows that the body is abandoning the contest; the subnormal temperature shows that the body has abandoned it." A wealth of evidence indicates that fever stimulates the reticulo-endothelial system to greater phagocytic action, that it increases the rate with which various immune bodies are formed and that it impairs the viability of organ-

isms in vivo as well as in vitro. Hyperthermia per se, by increasing the metabolic activity of cells increases the resistance to infection; the natural physiologic responses invoked (increased blood concentration, lessened oxygen-carrying capacity of the hemoglobin, increases in basal metabolism, rate and depth of respiration, rate of heart beat and various forms of cerebral irritation) are adequate to care for the increased oxygen requirements and acid products of metabolism (Wiggers).

Local signs vary with the nature of the wound, if any, and may be absent in desperate cases. Lymphangitis and lymphadenitis near the wound is common. Pain and vomiting are often ushered early. The pulse is weak and irregular: the heart rate is increased and hemic murmurs are often present. A diffuse erythematous rash with a dry, hot skin, is often seen in the early stages. The appearance of a cold clammy skin may portend a decline in toxemia or lessened resistance on the part of the patient. Constipation is the rule, offensive septic diarrhoea the exception. Infection of the renal epithelium is present to a variable extent. Delirium is the rule. Leucocytosis, as fever, is an index of the patient's resistance; a leucopenia presages a fatal outcome. Abscess formation, single or multiple, is a good sign, indicating localization of the focus or foci. The special signs and symptoms in gas gangrene are treated separately.

Treatment

Removal, diversion or drainage of the focus of infection before septicemia has manifested itself is the prime desideratum. Avoid or minimize all predisposing factors. Surgical judgment should determine how much a given patient can stand in the course of immediate or primary treatment. Many catastrophies will be avoided if first-aid is limited to checking of hemorrhage, immobilization of fractures or extensive lacerations and the avoidance of primary closure of lacerated wounds or amputations for crushing bone injuries. The relative importance of the associated shock must be evaluated before surgical treatment is undertaken. The immediate treatment of visceral lesions is discussed in connection with hemorrhage.

Up to recent years the results obtained by different kinds of treatment were unsatisfactory. Vaccines and sera have been employed with varying degree of success. With the introduction of sulfa drugs and particularly penicillin, the results obtained were greatly improved. Penicillin was found to be very effective in the treatment of staphylococcic, gonococcic, pneumococcic and hemolytic streptococcic infections.

Secondary (retarded) and not primary closure of deeply lacerated wounds, ample drainage, immobilization of fractures and gentle, rapid surgery will remain the prophylactic anchors in the prevention of septicemia.

GAS GANGRENE

No type of septicemia presents such a dramatic or fulminating clinical picture as does one of gas gangrene, once the exotoxins have entered the general circulation. During the World war I, even after military surgeons had learned, often through bitter experiences, to recognize its earliest manifestations, the total mortality of gas gangrene cases was 48.52 per cent. As might be expected, the comparative rarity of gas gangrene infestations in civil practice has rendered it difficult for the average practitioner to make early diagnoses, hence the mortality is higher than pertained in military surgery. Miller, Eliason, Collander and others estimate the mortality rate to lie somewhere between 49.7 and 75 per cent, the latter high figure pertaining to amputations for combined diabetic gangrene and gas bacillus infection.

While no one anaërobe can be said to be responsible for the infection, the Welch bacillus (*bacillus aërogenes capsulatus* or *clostridium Welchii*), can be cultured from cases of gas gangrene in at least 80 per cent of all cases. Anaërobes inhabit the soil, alimentary tract of man and animals, dairy products and clothing. These organisms give rise to exotoxins which act as aggressins. In the presence of such exotoxins anaërobes take on a virulent pathogenic character and rapidly lead to gangrene and death. In the late weeks of the World war I we saw fulminating cases of gas gangrene developed 4-6 hours after the soldier had received his wound or wounds. The most important factor in the production of gas gangrene is the intensive damage to muscle at a distance from the surface, under conditions that prevent the free access of oxygen. Next in etiologic importance is the local ischemia produced by the damage to the circulation. Deeply lacerated wounds, especially those involving muscle groups, contused, soiled wounds, compound fractures, hematomata and interference with the blood supply create favorable conditions for the growth of anaërobes. Gunshot, shell fragments or any punctured wound into which anaërobes are introduced by contaminated missiles, bits of clothing or injuries to the gluteal and perineal regions where fecal contamination readily occurs, all predispose to gas gangrene. Rare causes are abortions, instrumental deliveries, hypodermic or intramuscular injections and amputations for arteriosclerotic or diabetic gangrene.

Symptoms

The earliest symptoms are: sudden lancinating pain at the site of the wound, a sense of constriction of the segment of the limb which has been dressed and too tightly bandaged, headache, rising fever and pulse rate followed shortly afterwards by constitutional symptoms of grave intoxication, i.e. restlessness, delirium, stupor and coma. Locally there is found a rapidly increasing swelling, a serous or sero-sanguineous dis-

charge having an odor variously described as ammoniacal, musty or nauseating, and a mottled discoloration of the skin. Percussion over the limb or muscle group may give a subdued tympanitic note several hours before crepitation can be elicited. Gentle palpation often elicits crepitus; a more delicate method is to place a stethoscope over the limb and squeeze the muscle or group of muscles involved—a crepitant sound may be heard which is not recognizable to the touch. The invaded muscles contract slowly and the range of contraction is less than in the normal. Occasionally there may be an escape of gas bubbles when the edges of the wound are spread. Early x-ray examination is capable of demonstrating gas in the depth of the tissues long before it can be demonstrated by palpation or percussion. Brilliant green stains devitalize muscles less rapidly and less characteristically than in the normal. The clinical types often merge rapidly from superficial to deep or from local to massive, yet one can recognize: (1) Gas cellulitis, (2) localized gangrene and (3) massive gangrene of an entire limb.

TYPE 1 (LOCAL, DIFFUSE). (a) *Local cellulitis.* There is a reddening of the skin, sometimes a bronzing or brick-red color. A characteristic foul odor emanates from the wound discharges with bubbles of gas in the discharge. There is no gas in the deeper tissues. A mild toxemia is present.

(b) *Diffuse cellulitis.* The skin becomes mottled with blue, purplish or gray blotches and frequent bronzing of the skin takes place. The limb is swollen and edematous; there is elicited a crackling sensation on touching the skin. The distal circulation is unaffected. There is a profound toxemia with rapid pulse of small volume, variable temperature, cold extremities and, often, hiccough and emesis.

TYPE 2 (LOCALIZED GANGRENE). Usually associated with a compound fracture, especially of the femur. The skin is mottled purple. Localized crackling of the skin and deeper tissues can easily be elicited. The limb is not uniformly swollen. A foul, dirty fluid, containing bubbles of gas, but no typical pus, exudes from the wound. Toxemia of variable degree is present. The distal circulation may show signs of obstruction.

TYPE 3 (MASSIVE GANGRENE). The onset may be extremely rapid, often occurring in a patient apparently well on the way to recovery. It usually develops within forty-eight to seventy-two hours after the receipt of the injury but has been known to appear within four to six hours. The most severe type is seen in those wounds where there has been a severe disturbance of circulation (torn artery with inadequate collateral circulation, crushing of the principal vein of a limb, prolonged use of a tourniquet). The process may be a progressive one from type one or two, or massive gangrene may develop without warning, invading an entire cross-section of a limb simultaneously.

Characteristic symptoms:

1. Sudden, sharp, lancinating pain in the limb, below the wound.
2. Marked edema of the limb, with blotchy, purplish discoloration of the skin.
3. Obliteration of the distal pulse.
4. Profound toxemia with high temperature and rapid, low tension pulse. Temperature tends to become subnormal a few hours before death.
5. Characteristic ammoniacal, musty, nauseating odor around the wound.
6. From the wound opening issues a scanty discharge composed of broken-down blood clot, of brownish color, containing bubbles of carbon-dioxide gas.

Examination of the wound will reveal the following picture:

1. The subcutaneous tissues are filled with lymph-like fluid containing bubbles of gas.
2. The intermuscular septa and muscle sheaths are similarly filled with lymph-like fluid and gas; there is often separation between sheath and muscle tissue, extending beyond the wound for a considerable distance.
3. Accumulations of fluid and gas are found around the shaft of the bone or bones involved.
4. The muscle tissues are terra-cotta colored and avascular, and portions of muscle have disintegrated into foul-smelling necrotic masses. There is extensive gas infiltration of the muscle fibers.
5. In consequence of the muscle changes, bands of fascia stand out prominently.
6. Gas tends to spread along the perivascular tissues and planes of intermuscular connective tissues.

Prognosis

1. AS REGARDS THE INTEGRITY OF THE LIMB. a) Prompt recognition and treatment of type No. 1 will usually save the limb.

b) In the localized gangrene type, the prognosis is fair, providing distal circulation can be restored and the wound rendered surgically clean and mechanically drained.

c) In the massive type of gangrene the limb cannot be saved.

2. AS REGARDS LIFE OF THE PATIENT. A sudden fall of temperature to subnormal, with rapid, shallow respirations, low blood pressure and cold extremities, indicates that the exotoxins are freely circulating in the blood. The mortality in such cases is 100 per cent. Early recognition of the massive type of gangrene with prompt, high, amputation, will save 50 per cent of these cases.

Treatment

1. A lacerated wound, especially if it involves deep muscle planes, which is kept wide open so that oxygen can come in contact with the bruised tissues, will not develop gas gangrene. Primary closure of such wounds is dangerous.

2. A sudden change in the clinical picture of a patient with a lacerated wound demands instant revision of the wound. Should crepitus and other suggestive signs be elicited reopen the wound if it has been sutured, separate the fascial planes between muscle bundles. The wound is dusted with 5 gm. of sulfathiazole or sulfanilamide. The patient then is given sulfanilamide or sulfathiazole orally—2 gm. as the first dose and then 1 gm. three times daily until the condition improves.

Before the advent of sulfa therapy and of penicillin, the chief therapeutic remedy was administration of antitoxins and also local application of zinc peroxide. The mortality observed after this treatment was very high. In the recent years this treatment was replaced by administration of sulfa drugs and penicillin. The mortality by the latter method of treatment (sulfa drugs and penicillin) is considerably reduced.

3. Delay bone repair, other than immobilization, and wound closure until all toxemia has disappeared.

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Chapter III

Anesthesia in Urgent Surgery

By A. M. DOGLIOTTI

It is well known that insensitivity in surgical operations may be achieved by several methods. In emergency surgery that method must be applied which is the safest, simplest and quickest. The following is a brief description of those procedures which are more frequently used, leaving to the textbooks* on anesthesia the discussion of the finer particulars and more complex technics.

The means available for anesthesia can be divided into two major groups: (1) **General anesthesia or narcosis:** By inhalation, through the venous route, *per rectum*, or by hypodermic injection. (2) **Peripheral anesthesia:** Local, regional, block and spinal. A separate group, supplementing the general and peripheral anesthesia, includes the so-called pre-anesthetics or base-narcotics which aid in diminishing the patient's general sensitivity, thus serving as a preparatory measure.

NARCOSIS

Narcosis is the administration of drugs the general action of which renders the patient insensitive to pain, inducing deep sleep for the duration of a surgical procedure. Besides deep sleep and insensitivity, there are other factors rendering a narcotic acceptable to surgery, i.e., muscular relaxation and suppression of the unconscious movements of defense. Furthermore, when narcosis is ended, former conditions must be restored without delay and with no secondary or dangerous effects upon the structures and functions of the body. Of those narcotics which have these prerequisites, the following are to be preferred: (1) those which have the most rapid action and are quickly eliminated; (2) those which are more agreeable to the patient; and (3) those which are easy to administer.

The last condition is important in emergency surgery, especially in war surgery. The surgeon at a First Aid station must invariably operate with limited assistance and without the equipment of modern anesthesia, and upon patients who are not in a condition to tolerate special preparation. Furthermore, he is confronted with the necessity of saving time so as to take care of several wounded who have arrived at the same time.

* A. M. Dogliotti, *Anesthesia—Narcosis, Local, Regional, Spinal*, S. B. Debour, Publishers, Chicago, 1939.

GENERAL PATHOPHYSIOLOGY OF NARCOSIS. It must be borne in mind that all narcotics diffusing in the body through the blood have a simultaneous action on all tissues which have a different specific resistance. The inhibition that first takes place, followed later by arrest of function, occurs in an elective way in the cells of the central nervous system and in the cells of the different parenchymatous structures as well as the other tissues. This, however, is a reversible action which for the ordinary dose does not cause appreciable damage to the vital organs of the vegetative system. Only in prolonged narcoses, which require large doses of narcotics, or in individuals with advanced visceral lesions, the damage caused by the narcotic may be considerable and sometimes very serious. *One should, therefore, abide by the simple but important rule to regulate the narcosis in such a way that it will not endanger the patient.*

So far as the action of the narcotic on the different centers of the nervous system is concerned it is known that they run in a course favorable to the surgeon's objectives. It is important that he bear in mind at all times the course of the narcosis and its possible complications.

The centers of the voluntary mobility and general sensitivity are the weaker. By increasing the concentration of the narcotic drug the function of the spinal and cerebellar centers is later arrested. When the dosage exceeds the so-called toxic limit, the bulbar centers of respiration and circulation are the last to be affected. The interval between the onset of muscular relaxation, the disappearance of reflexes and the first signs of damage to the bulbar centers of respiration and circulation is known as the "useful interval" or the "security margin." A satisfactory narcosis must take place at the inferior and not the superior limits of this interval.

Stages of Narcosis

A typical narcosis is differentiated into four stages: (1) mental stupor and relative analgesia; (2) motor excitation and deep analgesia (cerebral stage); (3) muscular relaxation and total anesthesia (spinal stage); and (4) return or period of awakening. The literature describes a stage of asphyxia, or bulbar stage, which follows the third stage. It is suggested that this stage is a complication of the narcosis, due to an excessive dose rather than a stage of the typical narcosis, and this state of asphyxia should never be reached under any circumstances.

THE FIRST STAGE begins when the first amount of the narcotic drug reaches the circulation. This stage is characterized by progressive psychic incoordination with increasingly deep change in external stimuli. The different types of physical sensitivity, especially that of pain, are weakened while the patient experiences abnormal auditory, visual and gustatory sensations. The higher mental functions are always more affected, and a recollection of events during this stage is vague, similar to the memory of a dream. On the other hand, there are no appreciable

changes in blood pressure, pulse, respiration, color of the skin, and the pupils, except in very apprehensive and sensitive individuals who were not given prenarotics.

IN THE SECOND STAGE consciousness is almost completely lost and, therefore, the patient does not, as a rule, remember anything from then on. There are some typical signs of motor excitement, more or less marked according to the type of patient, administration of prenarotics, and to the kind of narcotic applied.

The patient's movements are uncontrolled; tonic contractions, especially of certain muscular groups, i.e., masticatory, dorsal and extensor muscles of the extremities are present. The patient frequently screams without any reason and complains and speaks confusedly. Respiration is irregular and there are deep respiratory movements alternating with short periods of dyspnea. The pulse becomes fast and blood pressure rises, especially with ether, gas or ethylene chloride; the cutaneous temperature increases, the pupils become slightly myotic and the tendon reflexes are weakened. There is a red congestive discoloration of the skin with ether and ethylene chloride, bright red coloring with acetylene and narcilene, and slight cyanosis with nitrogen protoxide.

THE THIRD STAGE or deep narcosis is characterized by complete suspension of all psychic activities, abolition of reflexes, complete physical insensibility, and considerable relaxation of the striated musculature. Only the function of the respiratory muscles persists although diminished in range. Respiration becomes more regular with an increase in frequency as compared to the initial phase. The pulse rate, which had increased in the stage of motor excitement, drops to almost normal limits. Blood pressure is decreased under narcosis with chloroform, avertin, and endovenously employed barbiturates; it remains at an almost normal level in ether narcosis and rises in narcosis with gases. The pupils become myotic and do not react to light in deep narcosis. Observation of the pupils is very important because a sudden dilation without a corneal reflex indicates imminent danger of paralysis of the bulbar centers. In such an event the mask must be removed immediately, and circulation as well as respiration must be stimulated. In very deep narcosis the conjunctival reflex disappears and the corneal reflex is very weak or absent. Absence of the corneal reflex indicates that the narcosis is close to the danger point and, therefore, further administration of the narcotic must be watched very carefully.

STAGE OF AWAKENING. When the narcotic is no longer administered, the body begins to eliminate it progressively, the time varying according to the type of narcotic employed. Upon observing the patient, after operation, a gradual return of the functions temporarily inhibited sets in: reappearance of the tendon reflexes, increased pulse rate, deepened respiration and, finally, a gradual return of all other forms of sensibility

and consciousness. Usually, there is a second stage of motor excitement and finally all effects of the narcosis subside.

Accidents Caused by Narcosis

Only the more common and dangerous accidents occurring with narcosis are mentioned here. Rare accidents and postoperative complications can be found in manuals on anesthesia.

CARDIOVASCULAR ACCIDENTS occur by the direct action of drugs upon the heart and nervous centers which regulate the circulation. These accidents may take place at any time during narcosis and are due chiefly to an excessive dose. Conditions of special weakness of the heart and nervous centers, congenital or acquired, such as shock, severe anemia, toxemia, visceral damage and the like, are the most frequent causes for accidents of this type.

SUDDEN ARREST OF THE HEART AT THE BEGINNING OF NARCOSIS. This is a very serious accident which is extremely rare since chloroform has been discarded. It practically never occurs with ether or gas. It may ensue in exceptional cases when ethylene chloride is employed or after intravenous administration of barbiturates; it is then due to large initial dose in the presence of a marked weakness of the bulbar centers. The mechanism of a reflex inhibition, following a severe irritative stimulus of the mucosa of the upper respiratory tract, is considered to have lost most of its former importance. The introduction of preanesthetics, which decrease the direct sensitivity and anxiety of the patient, has proved very useful in preventing such a reflex mechanism. Furthermore, the general practice is to begin the narcosis with a very small initial dose and to increase it gradually.

ARREST OF THE HEART IN ADVANCED STAGES OF NARCOSIS. This condition is usually due to an excessive dose. This accident will not occur if the anesthetist observes the patient carefully and increases the dose of the narcotic with caution. As a rule, the heart stops after the arrest of respiration, and there are warning signs of cardiovascular insufficiency, such as small pulse, low blood pressure, pallor and marked cyanosis. The heart may stop beating before respiration ceases. In such a case the anesthetist has to recognize the danger and take the necessary measures. The narcosis must be stopped at once, artificial respiration given and carbogen (oxygen plus 5 per cent CO_2) administered. Coramine or caffeine may be injected intravenously. In serious cases, $\frac{1}{4}$ mg. of adrenalin is injected intravenously while artificial respiration is maintained and the precordial region vigorously massaged. If after two to three minutes the heart is still not beating, more potent measures have to be employed, such as intracardiac puncture and direct massage of the heart.

INTRACARDIAC INJECTION. With this method the life of the patient is sometimes saved, and it should be employed in every case of arrested

heart beat due to narcosis. To render this method successful intracardiac injection should be performed not later than five minutes after the heart has stopped beating. While artificial respiration and massage of the precordial region is maintained by one assistant and the intravenous injection is given, another assistant should prepare the equipment for an intracardiac injection. This consists of a thin, long needle to puncture the right ventricle and two syringes, one of which is filled with veritol or caffeine, the other with 5 cc. of physiologic solution and $\frac{1}{2}$ cc. of 1 per cent solution of adrenalin. The elective point for the injection is the fourth intercostal space, close to the costal margin. The needle must be introduced 3 to 4, sometimes 5 cm. in depth and in a slightly oblique direction toward the median line. The needle must be applied quickly and forcibly to overcome the resistance of the cardiac muscle. The first syringe with caffeine and veritol is injected as soon as blood flows from the needle which is left in place while artificial respiration is continued. If after twenty to thirty seconds the heart beats are not restored, the second syringe (diluted solution of adrenalin) is injected. Then the needle is removed while artificial respiration is maintained with rhythmic, strong compressions of the chest.

DIRECT MASSAGE OF THE HEART may be applied as a last resort in abdominal operations after the peritoneum has already been opened. In such a case the surgeon introduces his hand into the abdominal cavity to reach the diaphragm which, being relaxed, will allow rhythmic compression of the heart. If the abdomen is not open, the outcome of a laparotomy is very doubtful and, therefore, the surgeon should limit himself to external maneuvers.

RESPIRATORY ACCIDENTS. During the first and second stages of narcosis laryngopharyngeal spasms and contractions of the respiratory muscles often take place. The former may be explained by the irritating action of the narcotic upon the upper respiratory tract, the latter by the same mechanism which produces spasms of other muscle groups. By preparing the patient with preanesthetics, administering the narcotic slowly, and keeping the respiratory passages free, the frequency and severity of these accidents may be greatly reduced.

The treatment consists of forcible elevation of the lower jaw and, if this proves ineffective, the introduction of a tube of the Mayo type into the pharynx.

Periods of apnea, sometimes lasting half a minute or longer, may be observed in the first phase of narcosis. This is due to a reflex respiratory inhibition or to a persistent spasm of the respiratory muscles. Usually the apnea is easily controlled by raising the jaw or exerting slight traction on the tongue after a gag has been applied. In some cases, it is convenient to slap the patient's face slightly with the hand or a wet towel. In more persistent cases the apnea can be controlled by rhythmic com-

pressions of the chest. Sometimes apnea follows acapnia which is caused by excessive pulmonary ventilation, as after a period of hyperpnea. In these cases respiration is resumed spontaneously as soon as the body has sufficient accumulation of carbon dioxide: it can be enhanced by letting the patient inhale carbogen ($O_2 + 5\% CO_2$).

A TRUE RESPIRATORY PARALYSIS may be due to an excessive dose at any period of narcosis. It rarely occurs at the onset when it is caused by an excessive initial administration. It is more frequent in the period of deep narcosis when it is obviously due to accumulation of the anesthetic. The respiratory paralysis has to be taken into consideration in extensive narcoses, in patients in a serious condition of shock, anemia or some form of toxemia. It is usually preceded by gradually reduced amplitude of the respiratory movements and, therefore, the careful anesthetist will prevent its occurrence. As a rule, it is sufficient to discontinue the anesthetic at the first signs of respiratory depression until respiration is gradually resumed. If this fails, stimulants like coramine, veritol and caffeine must be given, and if the respiratory function is still reduced or completely suspended, artificial respiration must be given without delay and, at the same time, a mixture of oxygen and carbon dioxide administered. Rhythmic tractions of the tongue and mechanical stimuli on the skin of the face also are very useful.

When the patient is lying on his back the best method to apply artificial respiration is to lower his head, introduce gag and tongue tractor to prevent the tongue from dropping into the pharynx, and then start rhythmic compressions on the arms alternating with chest compressions. By grasping the wrists of the patient his arms are brought upward to the side of the head, pulling them energetically for one or two seconds; then the arms are brought back anteriorly and in a flexed position against the chest, thus compressing it to achieve a complete expiration. About ten to twelve complete movements should be performed in one minute, such movements being calm and even. At the same time carbogen and stimulants like lobeline, coramine, veritol or caffeine are administered.

Generally, with this technic complete pulmonary ventilation is attained and, within a few minutes, sufficient narcotic is eliminated to re-establish respiration. In more persistent cases, especially when there is little possibility for good thoracic expansion, active artificial respiration must be given while direct insufflation of a mixture of O_2 and CO_2 into the respiratory system is carried out.

This is best achieved by rhythmic insufflation into the trachea. However, the necessary instruments for tracheal intubation must be employed. If such instruments are not available, the mixture of gases may be insufflated, rhythmically and under pressure, into the mouth or through the nose, keeping these openings well closed and the stomach

compressed in order to prevent the passing of the gas through the esophagus. With an active distention of the chest under moderate pressure 20 to 30 mm. Hg. spontaneous respiration is attained through the elasticity of the chest. In cases of tracheal obstruction due to tracheal softening, direct insufflation into the trachea is indicated by means of a tracheotomy opening.

Technic of Narcosis

NARCOSIS BY INHALATION. This is achieved by inhalation of narcotic gases in a gradually increasing concentration, until the desired result is obtained. The narcotic passes into the general circulation because of its solubility in the blood, circulating through the capillaries of the pulmonary alveoli. When administration of the narcotic is suspended it is eliminated principally by the same route and only a small part escapes through other organs. The more commonly employed narcotics for inhalation are: ether, ethyl chloride, the narcotic gases, i.e., protoxide of nitrogen, ethylene, narcilene and cyclopropane. Chloroform is very rarely used because it is highly toxic. However, some surgeons still use it mixed with ether and ether chloride.

Ether. Ethylic ether or ethyl oxide ($C_2H_5)_2O$ is a colorless, clear fluid, with a characteristic odor. It easily dissolves fats, is highly inflammable, and has a boiling point of 98° to $100^{\circ}C$. With the use of closed masks, however, volatilization of this gas in the operating room is slight and the risk of fire or explosion is virtually non-existent.

Methods. The oldest method of administering gas is with the open mask of the Esmarch-Schimmelbusch type. An impermeable cloth or a wet towel must be placed upon it to limit dispersion of the gas. This method requires a large amount of ether to obtain the required concentration of gas in the inspired air and is now, therefore, used only for children for whom it is not advisable to limit the concentration of oxygen while it is dangerous to have an accumulation of carbon dioxide.

In cases of adult patients, it is advisable to use any apparatus suitable for mixed narcosis or a semi-closed adjustable mask of the well known type of Ombrédanne. The author improved upon this type with the following advance over the original mask of Ombrédanne: (1) ether, narcotic mixtures, ethyl chloride and narcotic gases may be administered separately or simultaneously; (2) oxygen and carbon dioxide may be given without moving the mask; (3) the filters which hold the ether may be easily changed and sterilized, thereby overcoming the disadvantages of the Ombrédanne mask which becomes moist after narcosis has been given, thus presenting some difficulty and also risk for later use. This improved apparatus is light and practical, and extensive experience on the part of the author has proved its superiority over many other types.

Technic of Ether Administration. When the open mask, such as described here, is used, it is covered with impermeable linen or paper, leaving a small opening in the center through which the ether is poured drop by drop. With a semi-closed mask or an apparatus for mixed narcosis the ether is given through a minute opening in the ether container. It is advisable to ask the patient to count slowly and loudly to divert his attention and regulate his respiration. Should defensive muscular spasm or voluntary apnea follow due to the irritative action of the ether, the amount of gas must be reduced by removing the mask for a few seconds. If the patient struggles considerably, narcosis should be started with ethyl chloride or gas until consciousness is lost and the period of agitation and spasm is initiated. Then the ether is increased, in our type or a similar mask, to the maximum graduations (4 to 5 in our mask).

As soon as muscular relaxation is attained the amount of ether is again reduced (2 to 3 in our mask) while carefully watching the pulse, respiration, color of the skin and ocular reflexes. When relaxation has reached its depth, the amount of ether is kept at its lowest level, although a return to the second period, which is characterized by contractions, especially of the abdominal and respiratory muscles, must be prevented. With our mask the narcosis is maintained with the pointer at 2.

When it is difficult to keep open the upper respiratory passages, Mayo's oropharyngeal tube may be introduced at the onset of the third stage. With our apparatus, the narcosis may at any time be quickly intensified by adding small amounts of ethylene chloride (pressing on the button close to the insertion of the small tube of the ethylene chloride). In the same way, awakening may be facilitated by bringing the indicator of the ether down to zero and allowing carbogen to flow into the mask through the special connection to which is attached the rubber tube of the carbogen container. Generally, the administration of ether ceases when the surgeon begins to suture the skin.

Ethyl-chloride, kylene, or ether ethyl-chloride (C_2H_5Cl) is a colorless, clear, inflammable fluid with a characteristic odor, and very fast evaporation, with its boiling point at 11° to $12^\circ C$. It is obtainable in hermetically sealed, small tubes. When used as a narcotic it is poured drop by drop on an ordinary Esmarch mask, or it is administered by an apparatus devised to facilitate application.

The narcotic action of ethyl chloride is as speedy as its elimination. This presents a great advantage but also a risk because of the toxic accumulation, especially in consideration of the small margin of tolerance between the period of deep narcosis and of the bulbar centers.

Administration with Open Masks. The Esmarch type of mask is covered with gauze and impermeable linen, leaving a central opening. The mask is applied to the face of the patient and a few drops of the

narcotic are poured into the opening. A minimal dose should be given at first to avoid defense contractions of the glottis and voluntary apnea. The patient is encouraged to talk and count. By gradually increasing the dose the patient loses consciousness after two to three minutes; the period of excitement sets in with marked contractions especially if pre-narcotics have not been administered. During this period many minor operations which do not involve exposure of the major cavities may be performed.

To achieve muscular relaxation the dose must be further increased, but it must be taken into consideration that ethyl chloride is not a suitable narcotic for this purpose, due to the small margin of safety between relaxation and the moment of danger of paralysis of the respiratory and circulatory centers. A skilled anesthetist, however, may obtain the best results by observing these rules: (1) Begin with a minimal dose; (2) allow at least five minutes for the onset of deep narcosis; (3) avoid cyanosis by allowing active oxygenation throughout the anesthesia. The patient awakens within a few minutes and toxic complications are minimal and of short duration. However, a copious tracheal secretion is quite a common occurrence, more so than with ether narcosis. This may be prevented by injecting atropin together with the pre-narcotic. Vomiting is frequent and usually subsides within one hour after the narcosis.

Nitrogen Oxide (Nitrous Oxide). This is an excellent narcotic for short anesthesia when complete muscular relaxation is not required. It is administered with one of the different types of apparatus for gas narcosis. The characteristics and use of these apparatuses are described in the textbooks on anesthesia. The best results are obtained by using pure nitrogen oxide with 10 per cent oxygen. This mixture acts quickly and complete anesthesia is attained within two minutes for a brief surgical operation. This anesthetic is eliminated with equal speed, and there are no disturbances afterward because the substance is virtually devoid of toxicity. It is safe to administer this narcotic even repeatedly.

Ethylene and Narcilene. These anesthetic gases, owing to their minimal toxicity, are well tolerated by patients even in a serious state. They may be administered as a mixture with 15 to 20 per cent oxygen or with ether, using one of the common types of apparatus for gas anesthesia.

Deep anesthesia is reached within eight to ten minutes and any major operation may be performed. For abdominal operations requiring complete relaxation, a small amount of ether is usually mixed with the gas. The gases are speedily eliminated without any damage to the organs or systems. Only an expert anesthetist can properly control an apparatus for mixed gas narcosis, thus attaining with safety the best results with a minimal amount of gas, with the use of the rebreathing method. There is, however, some danger of explosion, and the anesthetist must prevent as much as possible dispersion of the gases. The surgeon must avoid the

use of an electric scalpel or a thermocautery close to the face of the patient.

Cyclopropane. In recent years this gas has taken the place of ethylene and narcilene, because it is equally free of toxic effects and has a quicker and deeper narcotic action with a much smaller concentration. While the concentration of nitrogen oxide should be 90 per cent, that of ethylene 80 to 85 per cent, and of narcilene 70 per cent, that of cyclopropane should be only 10 per cent to achieve corresponding results. It is only with a concentration of more than 30 per cent that any risk to cardiac function and of renal damage become manifest.

Narcotic Mixtures and Mixed Narcosis. An expert anesthetist uses mixed narcotics if the necessary equipment is at his disposal. It is especially in emergency surgery, when it becomes necessary to start the narcosis quickly, that ethyl chloride and nitrous oxide are used with ether or other gases. At times, particularly in some European countries, mixture of liquid narcotics are employed to derive all the advantages of the different components without having to reach a dangerous dose of any one gas. Practically, however, the results are no better than when one uses only the less toxic narcotics of the mixture. Ether is contained in almost all mixtures, and the author believes that pure ether anesthesia is, as a rule, superior to the different mixtures.

While on the subject of mixtures, there is disagreement as to their relative value. The following are the most frequently used: (1) Billroth's mixture: one part of alcohol, three parts of ether, one part of chloroform; (2) A. B. C. mixture of the English: one part of alcohol, three parts of ether, two parts of chloroform; (3) Schleich's mixture: ether 60, chloroform 20, ethylene chloride 10 parts. Similar mixtures are those of Wulfig and Pelot.

NARCOSIS PER RECTUM. This type of narcosis is not frequently employed in emergency surgery and is, therefore, not described at length in this discussion. Narcosis per rectum requires a special preparation of the patient and more time to achieve the necessary results. Moreover, the patient has to be under careful observation owing to slow awakening.

The following is a brief description of the method which can be readily employed, provided enough time is available and the operator has experience in its administration and control:

NARCOSIS WITH ETHER OIL. On the day prior to the operation the patient is given an enema because the absorbing power of the large bowel is not affected as it would be if the enema were administered on the day of the operation. However, the enema is not essential. The patient is given one of the general pre-anesthetics as in other methods of anesthesia. A formula is prepared as follows: pure olive oil, 1 gm. per kg. of body weight, pure ether for narcosis, 2 gm., per kg. of body weight; in other words, for a patient weighing 70 kg. mix 140 to 150 gm. of ether

with 70 to 80 gm. of oil. This dose may be slightly decreased or increased according to the patient's general state of resistance. An ordinary rectal tube or preferably one suitable for proctoclysis with an enlarged tip at its end is used. The mixture is slowly injected into the rectum and is administered in two doses in intervals of ten minutes; four to five minutes should be allowed for each dose. The tube is clamped and kept in place during the interval between the two injections and remains in the rectum after the last dose has been given. Absorption through the mucosa of the descending colon, the sigmoid and rectum proceeds gradually. After fifteen to twenty minutes narcosis sets in slowly and smoothly. The stage of excitement is usually short and slight. Anesthesia persists about one hour. It is not advisable to increase the dose when the narcosis is not sufficiently deep and muscular relaxation is not achieved. Small amounts of ether may be administered by inhalation. In this way the narcotic effects may be enhanced without danger to the patient. On the other hand, the narcosis can be checked when an overdose becomes evident. Awakening takes place gradually within about two hours.

When there are signs of overdose, i.e. circulatory and respiratory depression, the clamp is released and an enema of cold salt solution is given to stimulate the respiratory function. As a matter of fact, the absorbed ether is eliminated principally through the lungs. For this reason, artificial respiration, an injection of lobeline and caffeine, and the administration of carbogen produce the desired result within a few minutes.

INTRAVENOUS (ENDO-VENOUS) NARCOSIS. With the introduction of the barbiturates intravenous anesthesia has acquired great importance. This type of narcosis is of special value in emergency surgery because it meets many requirements, such as the need for operating with little assistance and in a short time. Intravenous narcosis does not necessitate special preparation of the patient except an intramuscular injection of one of the usual pre-anesthetics one-half to one hour prior to operation. Some pre-anesthetics, such as pantopon, may be administered intravenously as late as ten minutes before the narcosis.

The narcotic acts so quickly that operation can be begun four to five minutes after intravenous injection. The onset of the narcosis causes the patient no unpleasant sensation or memory. Furthermore, patients in a poor general condition tolerate this type of narcosis unless they have advanced hepatic damage or marked hypotension.

Awakening takes place rapidly so that twenty to thirty minutes after the narcosis is ended the patient does not require any supervision. The technic is simple, consisting of the slow and continuous intravenous injection of the drug during operation. Any physician, even without experience in anesthesia, may perform the injection under the supervision of a surgeon. The course of the anesthesia is similar to that of narcosis by inhalation. Sometimes, especially with eüpnarcon, there is a more

pronounced period of excitement prior to deep narcosis with relaxation.

This type of narcosis is indicated in almost all superficial operations, especially of the face and neck when an inhalation mask would be disturbing. It is less desirable for those abdominal procedures which necessitate complete relaxation which is not always attained with the prescribed dose. It is contraindicated in operations which last more than forty to sixty minutes, and for patients with advanced hepatic and renal damage or with marked hypotension.

Sodium Evipan (Evipal). Sodium evipan, or N-methyl-C-C-cicloesnil-methyl-malonil-urea is the popular barbiturate for intravenous use and quick action. It can be obtained in a fixed 10 per cent solution. The technic of the intravenous injection is the usual. The syringe is filled with 10 cc. of the solution, equal to 1 gm. of the substance, and the injection is made as follows: (1) The solution is injected very slowly at first so that the first 1 to 2 cc. of the solution takes one to two minutes; then the speed of the injection is increased and 1 cc. is injected every thirty seconds. The patient is watched as in any other form of narcosis. In a patient of normal size and resistance, 5 to 7 cc. is usually adequate to permit the beginning of an operation. (2) One-half to 1 cc. is injected each time, repeating every two to three or more minutes, according to the progress of the narcosis. The anesthetist has to regulate the dose according to the state of the narcosis. It has to be borne in mind that, as for all other narcotics, a set dose does not exist. There is no definite number of cubic centimeters to obtain identical results, the amount varies with the patient. In each case the depth and length of the narcosis may be increased or decreased by a greater or lesser amount of the narcotic. The drug disintegrates very rapidly in the body, especially in the liver and when the injection is discontinued the symptoms of narcosis soon disappear. (3) The maximum dose for a medium sized and resistant patient ranges safely between 10 to 20 cc. (1 to 2 gm. of the drug). Larger doses may be injected for prolonged narcosis but the operator must guard against depression of the circulation and respiration. With large doses there is a longer period of depression after the operation, due to the slow and difficult disintegration of the drug and cumulative phenomena.

Eunarcon, narcotene, endonarco, and like preparations are narcotics with effects similar to those of evipan. The technic of their administration is the same; the results show only slight variations.

PERIPHERAL ANESTHESIA

This group comprises all methods involving temporary interruption of the function of the nervous fibers at any point between their terminal ends and the nervous centers. This is achieved by injecting anesthetic solutions in the following way: (1) At the place of the terminal ends of

the nerve, in the area of the operation and the surrounding region: local and regional anesthesia. (2) Around the nerves that innervate the region of the operation: troncular or block anesthesia (Reclus-Braun method). (3) At the region of the large nervous plexuses: plexic and *paravertebral anesthesia*. (4) At the level of the nerves and spinal ganglia in the extradural space: *peridural segmentary anesthesia* (Dogliotti's method) and *epidural sacral anesthesia* (Corning-Bier method).

Peripheral forms of anesthesia which act on the mixed nerves usually produce at the same time block of the sensory fibers (*true analgesia*) and of the motor fibers (*akinesia*), leading, therefore, to insensitivity and muscular relaxation of the groups innervated by the anesthetized nervous branches. The more centrally the nerves are blocked, the more nervous branches will be involved; therefore, muscular relaxation will be more marked.

The chief advantage of peripheral anesthesia lies in the possibility of limiting the action of the drug to one part of the body. In this way the nervous centers are not endangered and the circulatory and respiratory systems and the large parenchymatous organs are not affected by the toxic action of the narcotic.

Many surgeons, including the author, have extended the use of peripheral anesthesia in normal surgery to such a degree that two-thirds of the operations are carried out under this type of anesthesia, limiting general narcosis to children and very nervous and anxious patients as a supplement to the incomplete peripheral anesthesia, and also in special cases for which general narcosis is more suitable.

In emergency surgery peripheral anesthesia, while important, is restricted because of the need for speed and the frequency of septic processes in which local or regional anesthesia is frequently contraindicated. However, some types of peripheral anesthesia may be valuable in emergency surgery, especially the subarachnoidal spinal anesthesia, in operations upon the kidneys, abdomen, pelvis and lower extremities, while local, direct anesthesia is suitable for closed reduction in fractures.

A very important factor in peripheral anesthesia is the employment of prenarcoics to aid the reduction of psychic trauma. Furthermore, consideration has to be given to the use of a light supplementary narcosis, such as ethyl chloride, gas, or the intravenous injection of barbiturates, when peripheral anesthesia proves inadequate. Thus the operation may be completed with a minimal amount of narcotics and at the same time the risks of narcosis are reduced to a minimum.

Anesthetics with Local Action and Their Dosages

COCAINE was the first substance used to obtain local anesthesia (Karl Koller, 1884). Today cocaine is limited to superficial applications in anesthesia of mucous membranes, especially in otorhinolaryngologic

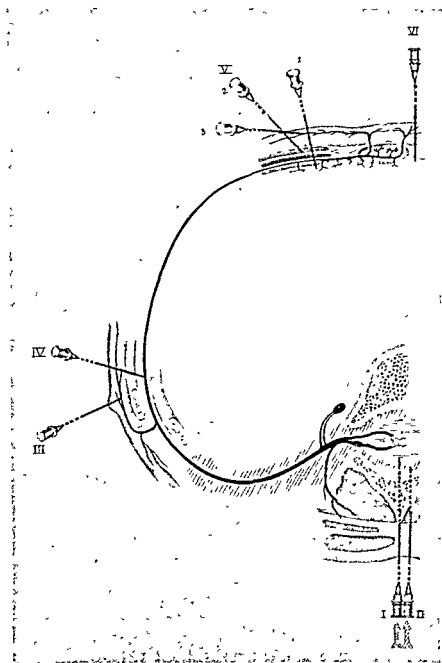


FIG. 31. Typical sites for injection of anesthetic solution. I. Subarachnoid spinal anesthesia. II. Peridural segmentary spinal anesthesia. III. Subcutaneous anesthesia. IV. Nerve-block anesthesia. V. Pararectus anesthesia: 1. properitoneal injection, 2. injection between the muscles of the abdomen, 3. injection into the subcutaneous tissues. VI. Midline local anesthesia.

practice. For this purpose very concentrated solutions are employed (5 to 10 per cent). The technic consists of the application of saturated gauze or cotton to the mucous surfaces for ten to fifteen minutes.

NOVOCAIN has an anesthetic power four to five times greater than

cocaine and is ten times less toxic. It is widely used in all forms of peripheral anesthesia in the following solutions and doses: (1) For local anesthesia by infiltration: aqueous solutions, $\frac{1}{2}$ to 1 per cent with 1 cc. adrenalin (1.0:1000.0) for every 100 cc. The maximal dose to be injected in a normal adult is 2 gm. of the substance, equivalent to 200 cc. of 1 per cent solution. (2) For nerve block, plexus or peridural anesthesia: 2 per cent aqueous solution with 1 cc. adrenal (1.0:1000.0) for every 100 cc. of the novocaine solution; maximum dose is 80 to 100 cc. (3) For subarachnoid anesthesia: dissolve 8 to 14 centigrams of the substance in the spinal fluid, or inject 2 to 3 cc. of a 4 per cent solution in distilled water without adding adrenalin.

PERCAIN AND PANTOCAIN have an anesthetic power ten times greater than that of novocaine and are four to five times more toxic. The doses are: (1) In local infiltration anesthesia: 0.2 per cent solution with 1 cc. adrenalin (1.0:1000.0) for every 100 cc.; maximal dose 100 to 150 cc. (2) Block, plexus and peridural anesthesia: 0.5 per cent solution with 1 cc. adrenalin (1.0:1000.0) for every 50 cc.; maximal dose 40 cc. (3) Subarachnoid spinal anesthesia: maximal dose 1 centigram to be dissolved in the spinal fluid, or in 2 to 3 cc. distilled water to obtain a solution of 0.4 per cent. The anesthetic action of percaine prevails about two hours.

General Remarks on the Pharmacologic Action of Local Anesthetics

The action of local anesthetics directly affects the nervous fibers that come in contact with the anesthetic solution by imbibition of the axis cylinder and not by swelling of the myelinic membrane as was believed until recently. Donaggio has shown that during the period of anesthesia the nervous fibers present evident morphologic changes which explain their interrupted function. This is a temporary and reversible lesion of which no trace may be found after the anesthetic solution has been absorbed and eliminated. However, these lesions may become permanent and cause the involved fiber to deteriorate if the anesthetic drug is allowed to act uninterruptedly for more than twenty-four hours (Malan).

The general action of local anesthetics is exerted through the circulation with phenomena of hypotension, slow respiration, dizziness and nausea. This, however, is not a pronounced action and alarming symptoms appear only in the event of overdosage administered to sensitive patients or those in poor general condition. No lasting appreciable effects have been apparent in the different organs or systems.

When vasoconstrictive substances, such as adrenalin, are added, the anesthetic action is noticeably enhanced, because these retard absorption in the circulation of the anesthetic solution. Thus an intensified and protracted action on the nervous fibers of the injected area is secured. Furthermore, the same degree of anesthesia may be achieved with a

lesser amount of anesthetic and, therefore, with less general toxic disturbances.

A further advantage in this combination is the possibility of attaining a temporary local ischemia which, within certain limits, facilitates the operation. A similar, though weaker, effect than in the case of adrenalin may be achieved with vasopressin, ephedrin and corbasil.

The use of adrenalin has become common in the loco-regional nerve block, plexus and peridural anesthetics. In anesthesia of large nervous trunks the use of adrenalin is essential because the anesthetic solution, injected in the vicinity of the nerve, penetrates it very slowly.

In subarachnoid spinal anesthesia the use of vasoconstrictor substances is dangerous and, therefore, inadvisable, especially since the secondary disturbances, i.e., headache, dizziness and radicular pain, are more intense. In the loco-regional type, anesthesia becomes apparent gradually after the solution is injected. It reaches its maximum about ten minutes after injection and lasts from forty-five minutes (novocaine) to one and one-half to two hours (percarine). In the nerve block, plexus and peridural anesthesia, narcosis appears to set in after fifteen to twenty minutes due to the penetration of the large nervous trunk; it lasts one to two hours, according to the solution used.

The dangers of local and regional anesthetics are essentially connected with the entrance of the solution into the circulation when a technical mistake has been made or the vitality of the injected tissue has been damaged. When the injection is erroneously made into a blood vessel, general disturbances like pallor, dizziness, nausea and vomiting, marked circulatory hypotension and choking sensation will appear immediately. Only when a very large amount of anesthetic is injected into the circulation will the patient's life be endangered due to bulbar paralysis with cessation of respiration and heart action. This, however, rarely occurs. Similar transitory disturbances of this type may be apparent in anesthesia of vascular tissues which absorb the anesthetic very rapidly; these are the neck, perineum, peridural and epidural spaces.

To prevent such complications the surgeon has to aspirate after each movement of the needle before the anesthetic is injected. If blood appears, the needle must be moved. In vascular tissues the solution must be injected very slowly. If disturbances occur, circulatory and respiratory stimulants must be injected and, if necessary, artificial respiration must be given with inhalation of carbogen.

When the solution containing adrenalin is injected into an artery, sudden ischemia may occur in the affected region and circumscribed areas may become necrotic.

As far as the local damage to the vitality of the injected tissues by the anesthetic is concerned, one should fear damage produced when the solution injected contains a high percentage of adrenalin. Many cases

have been reported of delayed healing of the surgical wound and even marginal necrosis; moreover, infectious, diffused processes have set in from the operative field to the injected area. Therefore, local anesthesia should be strictly avoided in operations of infected tissues.

General Rules for Peripheral Anesthetics

The instruments required for peripheral anesthesia are simple and may be reduced to a syringe and some fine needles. However, it is advisable to use a 10 to 20 cc. syringe of record type, equipped with three rings which make possible a more exact technic. A series of needles with "bayonet" attachments to the syringe is likewise useful. The needles are of different length and must be thin, unbreakable, sharp and have short points. It is important to keep the needles sharp so as to reduce the patient's pain. Moreover, the needles must be thin so that possible pricking of vessels or organs may prevent additional risks. The anesthetic solutions may be obtained in sterile vials or may be prepared for each administration by dissolving the anesthetic substance (tablets or powder) in physiologic solution. The latter may be sterilized by boiling; adrenalin is added immediately before use. The following table lists the more commonly used anesthetic solutions:

Solutions for Loco-regional Anesthetics

	Maximal Dose:
Novocain 0.5-1.0% + 1 cc. adrenalin (1.0:1,000.0) per 100 cc. solution	300 cc.
Stovaine 0.5-1.0% + 1 cc. adrenalin (1.0:1,000.0) per 100 cc. solution	150 cc.
Percaine 0.1-0.2% + 1 cc. adrenalin (1.0:1,000.0) per 100 cc. solution	150 cc.
Pantocain 0.1-0.2% + 1 cc. adrenalin (1.0:1000.0) per 100 cc. solution	60 cc.

Solutions for Nerve Block, Plexus and Peridural Anesthetics

Novocain 3% + 1 cc. adrenalin (1.0:1000.0) per 50 cc. solution	50 cc.
Percaine 0.4% + 1 cc. adrenalin (1.0:1000.0) per 50 cc. solution	30 cc.
Triplocaine	30 cc.

The vials of triplocaine, as used by the author, for peridural anesthesia, have the following composition: percaine 0.04 gm., pantocain 0.04 gm., novocain 0.10 gm., ephedrine 0.50 cc., adrenalin (1.0:1000.0) 0.5 cc., sodium chloride 0.2 gm., distilled water 30 cm.

Solutions for Spinal Subarachnoid Anesthesia

Novocain	10-14 Centigrams
Stovaine	8-10 Centigrams
Tutocain	4- 8 Centigrams
Tropocaine	4- 8 Centigrams
Percaine and Pantocain	1 Centigram

Solutions for Anesthesia of the Mucous Surfaces (Contact Anesthesia)

Cocaine 5-10%	10 cc.
Percaïne and pantocain 2% (in otorhinolaryngology)	10 cc.
Percaïne and pantocain 0.5% (in urology)	20 cc.

TECHNIC OF LOCO-REGIONAL ANESTHESIA. The anesthesia may be carried out on the operating table or in the preparatory room and all rules for asepsis employed. The anesthetist should wear sterile rubber gloves when preparing the region to be anesthetized by disinfecting the skin with tincture of iodine or mercurochrome and placing sterile towels around the area. It is always advisable at first to carry out an intradermic anesthesia, employing a fine needle, at those cutaneous points where the larger and longer needles will be introduced. The intradermic anesthesia is achieved by injecting some drops of anesthetic solution subdermally until a small white vesicle is formed. This will prevent or minimize pain when the needles are introduced and superficial points for orientation are established.

Then, the longer needles are introduced and the solution is injected into the deeper layers of the region. Next, the solution is injected along the line of the surgical incision. Special care must be taken so that the solution is distributed close to the derma. It is advisable to inject the anesthetic in the areas adjacent to the incision line and at a distance of 5 to 6 cm. from the operative field. Prior to injecting the solution, especially in anesthesia of the deeper layers and when operating in richly vascular regions, aspiration is indicated to avoid injecting the solution into a blood vessel.

The aspiration has to be repeated after each movement of the needle. Furthermore, the anesthetist must prevent the needle point from touching bony surfaces, because of their great periosteal sensitivity and also to avoid bending the needle point, i.e., curving the needle into a hook, which may cause damage to the tissues in the subsequent movements. When the deep layers of the tissues have to be injected in several points, the needle must be withdrawn each time to the subcutaneous layer and re-introduced in the required direction. An attempt to move the needle in different directions without first withdrawing it will increase the risk of breaking the needle; moreover, the needle can be moved only to a limited extent if not first removed.

COMMON TYPES OF PERIPHERAL ANESTHESIA. The different textbooks on anesthesia present detailed descriptions of anesthetics in different regions. The anesthetist has to acquire thorough knowledge of topographic anatomy and must consider the surgical requirements of operations. The author confines himself here to the more important characteristics of peripheral anesthesia.

LOCAL ANESTHESIA BY INFILTRATION:

Nerve block anesthesia

Anesthesia of the large nervous plexus

Peridural segmentary anesthesia (Dogliotti)

Epidural sacral anesthesia (Cathelin)

Subarachnoid spinal anesthesia (Corning-Bier)

Local anesthesia by infiltration is performed by injecting the anesthetic solution into the tissues which have to be crossed by the surgical incision. It is the simplest form and is used to remove a foreign body or a small subcutaneous tumor. One or more intradermal wheals are made, then the solution is injected subcutaneously following the line of the surgical incision.

If the intervention is to be deeper or the tumor to be removed is larger, the solution has to be injected also into the subfascial layers and around the area which will be operated upon. The infiltration of the anesthetic is, therefore, of the "cup" or "fan" type to block all nerves which innervate this region. To demonstrate such procedure we describe as an example local anesthesia as administered for an operation of inguinal hernia. In this operation a local subcutaneous and subfascial infiltration is made, also in the inguinal canal, along the line of incision; a "fan" infiltration is made above this area 2 to 3 cm. medially to the anterior iliac spine to create a subcutaneous, subfascial, intramuscular and preperitoneal anesthetic block at this level. A further example to demonstrate the frequent and suitable application in emergency surgery is the local anesthesia of the focus of fracture. The injection of 20 to 30 cc. of an anesthetic solution into the hematoma around the fracture makes possible a perfectly painless reduction (Lerda method).

Regional Block. In some cases it becomes necessary to produce muscular relaxation in a large region adjoining the immediate operative field; in such cases the solution has to be injected fan-wise at a certain distance bordering the region of operation. For example, for a midline laparotomy, which requires large exploratory movements, it will be necessary to use local anesthesia along the incision line and supplement it with a subcostal block on both sides in a median laparotomy and on one side in a lateral laparotomy.

Nerve Block Anesthesia. In some cases it is sufficient to block some nervous branches at some distance from the operative field. A nerve block is sometimes indicated because local infiltration of the operative field is contraindicated due to infectious processes. The classical example is nerve block anesthesia of the mandibular nerve at the Spix spine for operations on the lower teeth. Also, nerve block anesthesia is widely used in one or more intercostal nerves for rib resections and thoracoplasty.

Anesthesia of the Large Nervous Plexuses. This is not very frequently

used; in emergency surgery it is applied in anesthesia of the brachial plexus in operations of the upper extremities. Twenty to 25 cc. of the solution is used for the plexus and nerve block anesthesia.

Spinal Anesthesia. In emergency surgery, in which a simple technic and speedy action are imperative, **subarachnoidal rachianesthesia** (Corning-Bier) is chiefly employed. It is known that this anesthesia is achieved by injecting the anesthetic solution into the subarachnoidal space, at the level of the third or fourth lumbar intervertebral space or lumbosacral space. The injection may also be made in upper spaces, i.e., to the seventh or eighth thoracic when a high rachianesthesia which covers the entire abdomen is required. The spinal cord, above the first intervertebral lumbar space, calls for special care when the needle is introduced in order to prevent an injury to the spinal cord. The technic of the injection is quite simple. The patient is invited to seat himself and bend forward. When this is not possible the patient has to lie on his side curving his back as much as possible, without the pelvis being inclined but symmetric to the vertebral column. The needles have to be strong, unbreakable, with thin and short but sharp points. After disinfecting the skin the exact position of the last three or four lumbar spinous processes are located by pressing firmly against the median line. The needle is then introduced in the center between two conspicuous spinal processes, exactly on the median line perpendicular to the skin surface. Some anesthetists avoid the interspinal ligament, introducing the needle 3 to 4 mm. laterally to the median line and slightly inclined toward it.

As a rule, this is unnecessary as the interspinal ligament is readily penetrated by the needle. When the needle passes through the yellow intervertebral ligaments, a diminished resistance is distinctly observed and the stylet may be removed from the needle. If no spinal fluid is discharged, the needle has to be introduced further without using the stylet. The resistance of the pia mater is frequently observed, and when this is overcome the spinal fluid appears.

In cases of kyphoscoliosis, with torsion of the vertebral column, the technic is more intricate. The extent of the torsion has to be approximately determined and the needle has to be introduced in an oblique direction. Despite this the injection is usually successful.

When the puncture is made above the second lumbar space the needle has to be introduced very slowly to avoid damage to the spinal cord; this is achieved by stopping the needle when the point has perforated the dura mater. It is imperative to remove the stylet from the needle when the point has passed through the yellow ligaments. This method enables the operator to observe at once when the needle has penetrated the dural sac and spinal fluid appears; thus the needle will not be introduced farther. After introduction of the needle, some cc. of spinal fluid

are allowed to escape prior to connecting the needle with the syringe which contains the anesthetic solution. Where an extensive rachianesthesia above the point of injection is required, 4 to 6 cc. of spinal fluid are aspirated and then slowly re-injected. If the anesthesia required is not extensive, only 1 to 2 cc. of spinal fluid are aspirated and then added to the 2 cc. of the anesthetic solution, thus bringing the volume of the injected fluid to 3 to 4 cc. When a thoracic rachianesthesia (7th to 8th space) is performed for operations of the upper abdomen or lower chest, only 2 cc. of anesthetic solution are injected. With this method a region of maximal anesthesia is confined to three to four roots above and three to four roots below the point of injection, thereby preventing the anesthetic from diffusing toward the cervical portion and cauda equina. Thus a paralysis of any muscles, excepting the intercostal, is prevented. Paralysis of the diaphragm, innervated by cervical branches, is likewise avoided. Furthermore, complete anesthesia of branches of the cauda equina will not take place, the area of vascular paralysis is reduced and, therefore, the risk of arterial hypotension is diminished.

When the anesthetic is injected the needle is removed, and the patient is placed on his back with his head elevated. After four to five minutes the head is slightly lowered by placing the bed in a moderate Trendelenburg-Nélaton position.

With the above described method the risk of bulbar ischemia is prevented. Bulbar ischemia is due to arterial hypotension which is always present, in a higher or lesser degree, after subarachnoid anesthesia, especially in extensive anesthesia of this type and in hypotensive patients. It is of great importance that the anesthetist follow this method closely, thus availing himself of a definite safeguard against the most dangerous complications of this type of anesthesia. When suspicious signs of hypotension appear (soft pulse, pallor, respiratory paresis) the patient has to be placed in a pronounced Trendelenburg position.

Immediately after the spinal injection, the anesthetist has to administer an injection of ephedrine; where it is necessary, other hypertensive circulatory stimulants have to be injected. The anesthesia sets in within five to six minutes and lasts about one hour with novocaine and stovaine; percaine and pantocain anesthesia takes effect after ten minutes and lasts about two hours. Frequently, there is nausea and efforts to vomit after fifteen to twenty minutes. To avoid this a preoperative injection of atropine proves effective; also an injection of ephedrine or other hypertensive and circulatory stimulants, as well as inhalations of carbogen ($O_2 + CO_2$), are useful.

When, despite all precautions, dangerous symptoms of respiratory paralysis appear, the Trendelenburg position is increased, artificial respiration applied, and carbogen inhalations and injections of hypertensive stimulants administered.

Among postoperative complications the more frequent and troublesome is headache which, as a rule, sets in twelve to twenty-four hours after operation, sometimes lasting for several days. This may be relieved with injections of gynergen or nicotinic acid, or the administration of pyramidon tablets. In rare cases the physician has to resort to spinal puncture in hypertensive forms, or inject distilled water intravenously in hypotensive forms. Generally, the headache increases when the patient raises trunk and head (hypotensive form) and, therefore, a horizontal position or lowered head are indicated. A further point, frequently overlooked, is to avoid the use of hot water bottles and other heat-producing appliances, to feet and legs immediately after operation. The anesthesia sometimes persists for some hours, the patient is unable to feel excessive heat and, therefore, serious and extensive burns may result.

PERIDURAL SEGMENTARY RACHIANESTHESIA (DOGLIOTTI METHOD). A detailed description of this method is presented in Dogliotti's "Anesthesia." It has important advantages over the subarachnoid rachianesthesia (Corning-Bier); it is frequently employed, especially in abdominal and renal surgery. The procedure is that of injecting an anesthetic solution of the nerve block type, e.g., 25 to 30 cc. novocain solution 3 per cent, plus 1 cc. adrenalin (1.0:1,000.0), into the extradural space of the vertebral canal. The fluid diffuses readily in the fatty peridural connective tissue, and infiltrates the spinal nerves along the intervertebral openings. Thus an extensive nerve block anesthesia is attained over eight to ten pairs of spinal nerves. Complete anesthesia and muscular relaxation appear after fifteen minutes lasting about two hours. This anesthesia is of the segmentary spinal type without diffusion of the anesthetic into the spinal fluid.

The method presents the following advantages: (1) no marked drop of blood pressure which usually is the case after subarachnoid rachianesthesia, due to vascular paralysis in a very extensive visceral area; (2) no risk of diffusion of the anesthetic toward the cervical segment, and less still toward the medulla; (3) no vomiting during operation; (4) no postoperative headache.

Technic. In this method the technic is more intricate than that in subarachnoid anesthesia because the anesthetic has to penetrate into the vertebral canal without perforating the dura mater. If the rules are strictly observed, the results are satisfactory, as has been demonstrated by many anesthetics performed by the author and other physicians.

The needle has to be thick and have a very short bevel. After the usual wheal of anesthesia has been made on the midline of the level of the chosen intervertebral space, the needle is introduced through the spinous ligament or immediately lateral to it. When the needle reaches the yellow intervertebral ligaments the stylet is removed and the needle

is connected to a small manometer, provided by a small U-shaped glass cannula and containing a small amount of sterile water. Then the patient is invited to bend forward, and the needle is inserted slowly through the yellow ligament. When the needle penetrates the yellow ligament, entering the peridural space, the manometer will point to a rather marked, sometimes very strong *negative* pressure. At this point the needle is in its proper place and must not be pushed in farther to prevent perforation of the meninges. In the latter case the manometer shows positive pressure.

The manometer is then removed and 8 to 10 cc. of anesthetic solution are injected gradually while the needle is left in place without moving the patient. After four to five minutes a sensitivity test is made by touching the buttocks and legs with a needle; perfect sensitivity must be present. This test serves as a precautionary measure to check if the anesthetic solution has not penetrated into the subarachnoidal space. If this were the case a very rapid deep anesthesia would set in, from the point of injection downward, as in subarachnoid rachianesthesia. When this test is complete the remaining 15 to 20 cc. of anesthetic solution are injected, the needle removed, and the patient is laid down. To achieve a maximal anesthesia fifteen minutes should be allowed to elapse.

The experienced anesthetist may employ the following technic in the place of a manometer: When the needle, without stylet, passes through the yellow ligaments a syringe, containing some cc. of anesthetic solution, is connected to it. While the needle is inserted slowly through the yellow ligaments with one hand, the operator applies continuous pressure over the plunger syringe with his other hand. Up to the moment when the needle enters the yellow ligament, there is a strong resistance to the injection of the anesthetic solution, due to the thickness of the ligaments. As soon as the needle penetrates into the peridural space the resistance to the needle suddenly subsides, and it seems to the anesthetist as if he injected the solution into empty space. When the syringe is removed a check has to be made whether blood or spinal fluid is discharged; also, the jugular veins have to be compressed to increase pressure. If no blood or fluid appears, the anesthetic solution may be safely injected in successive doses, as previously described.

For surgery of the lower abdomen, below the level of the umbilicus, the injection is made into one of the intervertebral spaces between the tenth thoracic and the first lumbar vertebrae.

For surgery of the kidneys and upper abdomen (stomach, liver, spleen), or for operations on the lower chest, the injection is made into one of the intervertebral spaces between the fifth and eighth thoracic vertebrae. In these high thoracic injections the needle has to be in a markedly oblique direction because of the position of spinal processes.

Chapter IV

Electrosurgery

By GUSTAVUS M. BLECH

It seems paradoxical that electrosurgical methods have become indispensable in the domains of neurologic and urologic surgery yet few general surgeons avail themselves of the advantages offered by this type of operative technic. This is not the proper place for a discussion of the underlying causes of such a regrettable situation; they have been set forth in the writer's monograph¹ which is available in most medical libraries. It must suffice here to point out that electrosurgery offers distinct advantages which merit utilization in many cases of an urgent character while the disadvantages, if such they may be called, refer only to the need of an armamentarium and familiarization with its applicatory technics. There are no inherent contraindications to electrosurgery as such, since, as will be shown, it is subjected to the same indications and contraindications that affect any surgical intervention.

Obviously, it is of some importance to precede a discussion of the concerned technical problems by a few general remarks, at least for the benefit of readers who are not thoroughly familiar with the nature and purpose of electrosurgical procedures.

DEFINITIONS. The term electrosurgery is restricted to operative procedures carried out with the so-called high frequency current. This current is an alternating current but distinct from the commercial current used for lighting therein that instead of having only 120 changes of polarity each second (hence the name "low frequency") the changes run into hundreds of thousands even a million times per second (hence the designation "high frequency").

In a generic sense any current used for surgical purposes, such as the destruction of a small skin growth or a hair follicle by a weak galvanic (direct) current could be grouped as electrosurgery, but it seems preferable to retain the term "electrolysis" because it describes the nature of the effect produced upon the structures to be destroyed.

HISTO-PHYSIOLOGIC EFFECTS OF THE HIGH FREQUENCY CURRENT. The commercial alternating current applied to the human body produces neuromuscular contractions and shock. Such a current when intensified in power (voltage) is destructive of life by what is commonly spoken of as electrocution. In contrast to it, the high frequency current

¹ Gustavus M. Blech, *Clinical Electrosurgery*. Oxford University Press, 1938.

There are available a large number of electrosurgical apparatus, large, medium size and small ones, stationary and portable ones. Some are equipped with a so-called spark-gap, which arrangement is assumed to give the best current for heavy (deep) electrocoagulation, others are equipped with electronic tubes which are supposed to yield a good current for fine electrodissection, while still others utilize both the spark-gap and tubes in one and the same apparatus. The surgeon is little concerned with the interior arrangement of high frequency apparatus, which bears a certain resemblance to that seen in radios. A serviceable, portable and comparatively inexpensive apparatus is shown in the accompanying illustration. It has a special equipment for treating patients with short wave diathermy and lighting a small ultraviolet lamp for local heliotherapy. The apparatus is provided with a complete set of electrodes and a foot switch for electrosurgery.

GENERAL TECHNIC. Those unfamiliar with electrosurgery will do well to obtain a large piece of raw meat, preferably one with fat and tendons, place it on the dispersing electrode and, after setting the apparatus and the foot switch, practice light and coagulation dissection and coagulation of the various structures. It should be borne in mind that the electrodes themselves do not divide the tissue but the current reaching their tips from within the body does the division or destruction of the structures, as the case may be. For dissection the cutting current is applied by holding the tip of the electrotome barely in touch with the skin or other structure and moving it along the line of incision the same as one moves a scalpel, but without pressure. In other words one should perform electrodissection much as a draftsman holds a pencil lightly to draw a very fine line over the paper. As the endogenic heat splits a layer, the tip of the instrument is again placed in very light contact with the next layer to be divided. It is improper to turn on the current while the tip is held at some distance from the tissues because even at a millimeter or two of space between the electrode tip and the tissues sparks will jump across, something that is best avoided. Therefore, one poses the tip of the electrotome close to the tissue and then presses the foot down upon the foot switch to "open" the current. The effect is instantaneous if the apparatus is properly set.

For coagulation the same technic is followed, except that contact with the end of the tip of the electrode, which is ball shaped or flat, like a small disc, is more intimate than in electrotomy.

The coagulating current can be utilized to seal small blood vessels by contact of the electrode with them. Instead of ligatures, it is necessary only to catch the severed end of a small blood vessel with forceps. When the hemostat has firmly grasped the vessel, the coagulating electrode is brought in contact with the forceps and the current turned on for a few

seconds. When the forceps is removed it will be seen that bleeding has stopped—the severed end having been sealed, as it were.

This method is not suitable for large blood vessels, as the volume of blood in the severed vessel may be heated sufficiently to burst the wall of the vessel by explosion. Large blood vessels should therefore be clamped and ligated as in classic surgery.

GENERAL INDICATIONS. The general indications for the employment of electrosurgical procedures can be deducted from their local and constitutional effects.

The principal consideration is the fact that even extensive procedures are followed by little if any shock. This characteristic has been explained in several ways. It has been held that the sealing effect of the high frequency current blunts the severed ends of nerves thereby inhibiting the reflexes. While in the early period after the introduction of electrosurgery the method has been described by zealots as bloodless, the fact remains that it seals only capillaries and minute blood vessels during coagulating dissection, so that there is a reduction of the amount of blood lost. Furthermore, as was already stated, ligation of blood vessels becomes necessary only in those of medium or large caliber. The current *per se* is sterile, so that endogenic infection is reduced to the minimum. Lymph spaces and lymph vessels are closed (sealed) by the coagulating effect of the current, so that inoculation with both vegetable (bacteria) or animal cells is virtually non-existent.

Postoperative pain is greatly lessened probably for the very reasons given for the reduction or absence of shock, and it is therefore not surprising to see patients who have undergone the ordeal of a major operation appear in fine spirits throughout their convalescence.

What then are the clinical indications in urgent cases?

We have the authority of Cushing that operations of the brain, notably the removal of tumors that in the past have proved most hazardous, became less risky since the utilization of the surgical high frequency current. This epochal revolution in neurosurgery is purely due to the fact that the excessive bleeding and shock connected with the classic extirpation of brain tumors is greatly reduced, if not obviated, by electrotomy and electrocoagulation. If one applies that experience to similar conditions elsewhere in the human body one can see its wide field of activity.

The sealing properties of the high frequency current of capillaries and lymph vessels early suggested its indication in the removal of malignant growths. But so far as urgent surgery is concerned electrosurgery will find its most effective usefulness in the management of surgical infections. To this should be added cases of trauma which has reduced the somatic resistance of the individuals. Patients suffering from anemia

and those whose bleeding time indicates that they might be a bad operative risk would be the ones selected for electro-surgery for the reason above stated.

CONTRAINDICATIONS. As was already stated there are no contraindications to electrosurgery as such, that is to say, any contraindication to classic surgery would naturally apply also to operations carried out with the high frequency current. But it should be considered that the conditions prevailing with elective surgery do not apply to urgent surgery. The very urgency for surgical intervention clearly shows that certain risks cannot be shirked or postponed if life is to be saved and, as was just stressed, one often will find in electrosurgical technics a means of bringing an operation to a safe termination when classic measures would lessen the chance for success.

There is, however, one objectionable feature connected with the practice of electrosurgery and that is the danger from exploding volatile gases used for inhalation anesthesia. An accidental sparking may cause death. For that matter the following instance may serve as an object lesson. A noted European surgeon was operating for a pulmonary fistula. The patient was under the influence of ether anesthesia. Thoughtlessly the surgeon applied a red hot Paquelin cautery to the fistulous opening and in an instant the lung was burned to a crisp.

With ordinary precautions, such as placing a wet sheet between the patient's head and the body proper, any electrosurgical operation can be carried out with safety even under ether narcosis. Naturally anesthetics like cyclopropane must not be even in the operating room let alone used for anesthesia. With this exception there are no contraindications.

Chapter V

Laparotomy

By JULIUS L. SPIVACK

The dictum that a surgeon who deliberately opens the abdomen should be able to meet any and all arising contingencies, holds good even more so in urgent laparotomies. In non-emergency cases the diagnosis usually is made before operation so that the surgeon can plan his acts because he knows what to expect and where to look for it. In emergency cases the surgeon often is unable to avail himself of many valuable laboratory tests either because they are contraindicated on account of the type of suspected pathologic process or delay beyond safety.

It is particularly in these seemingly acute abdominal conditions that laboratory tests would be of great aid, as there are conditions outside the abdominal cavity that can give pictures strikingly resembling those of an "acute abdomen."

One cannot sufficiently stress the fallacy of the assumption that a wrong diagnosis of an abdominal condition is not calamitous because in any case the abdominal cavity has to be opened. It should be borne in mind that when the wrong viscus is suspected the incision may be too far away and the operative field improperly exposed. Indeed, one may not even be able to remedy the situation by increasing the length of the abdominal incision, so that at last another and more appropriate incision has to be made—by no means a happy solution of the problem. The situation becomes a tragedy when an extra-abdominal process is erroneously diagnosed as an acute intra-abdominal affection. Among these conditions in the first place should be mentioned coronary thrombosis, lobar pneumonia and pericarditis. The life of these patients is certainly endangered by the unnecessary laparotomy.

Among other conditions which have been mistaken for acute abdominal conditions should be mentioned renal ectopia, hydronephrosis and ureteral stones. Occasionally lesions of the central nervous system, such as tabes dorsalis, subarachnoid hemorrhage, celiac neurosis may simulate an acute abdominal process. Endocrine disturbances such as hyperthyroidism, hypothyroidism, hypoparathyroidism, ovarian affections or diabetes may closely resemble a picture of acute abdominal trouble. Systemic diseases, infectious or non-infectious, such as Henoch's purpura, abdominal allergy, malaria, undulant fever, tularemia, were occasionally confused with acute abdominal conditions. Patients suffering from rupture of the m. rectus, hematoma of the abdominal wall due to

spontaneous rupture of the inferior epigastric artery also have been unnecessarily subjected to laparotomy.

While in many cases a correct diagnosis can be made because the characteristic features of the particular disease will be manifest in addition to the confusing symptoms, others have been reported in which it was impossible to make a correct diagnosis and the true nature of the disease was discovered during the subsequent course of the disease or at necropsy.

A reliable sign which may be very helpful in differentiating between intra-abdominal and extra-abdominal condition is that in the former the patient prefers to lie flat on his back with the legs bent in the knees and hip-joints, while in chest conditions they prefer to take the half sitting posture. Another helpful sign is that in all cases of a perforated viscus the patient prefers to lie still, while in extraperitoneal conditions he is restless and moves from one position to another.

However, as valuable as these signs are, they do not always follow this pattern and solely to rely upon them may lead to disaster.

After the diagnosis of an acute abdominal condition is made one has to decide as to the site and type of the abdominal incision.

Abdominal Incisions

In opening the abdomen the following considerations have to be taken into account:

1. **THE INCISION SHOULD AFFORD A GOOD OPERATIVE FIELD.** It should therefore be of adequate length; a short incision impedes the work. If one has to err, it is better to make too long than too short an incision. For all operations in which a good view is essential, the length of the incision should range between $4\frac{1}{2}$ and 6 inches. For cases involving the removal of small tubular structure, such as the appendix or a fallopian tube the incision may be considerably smaller.

2. **THE INCISION SHOULD CAUSE NO SERIOUS DAMAGE TO THE ABDOMINAL WALL.** This is of great surgical importance. If the abdominal wall is damaged, several possibilities may arise:

a. The abdominal wall may give way in the line of incision at the time immediately following operation, producing disruption, with or without evisceration of the abdominal contents. Evisceration is responsible for a high mortality rate.

b. The abdominal wall may give way many months or even years later, producing an incisional hernia. In these cases adhesions are formed, producing pain and morbidity, and in many cases are the cause of internal strangulation of the bowel.

c. The muscles of the abdominal wall become atrophied, thus diminishing normal tonus of the abdominal wall; the viscera are not held to-

gether pressing against each other but are rather loose and the patient needs an abdominal support to keep them in place.

It is extremely difficult to avoid damaging the anterior abdominal wall, owing to the peculiar arrangement of its constituent parts. The topographic arrangement of the component parts of the abdominal wall

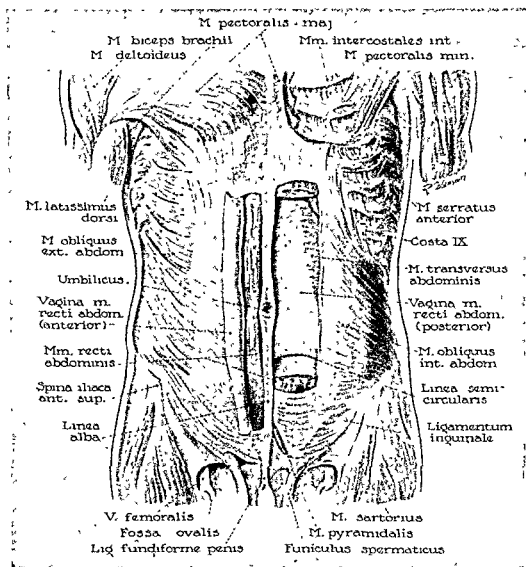


FIG. 33. Anatomy of the anterior abdominal wall.

is such that it offers good support for rather movable structures (intestines) suspended on long attachments (mesentery) in spite of the erect posture; in fact, there is no place in the abdominal wall where a large opening can be made without damaging it. In order to make this clear, a brief topographico-anatomical description is not amiss (Figs. 33, 34, 35).

The layers of abdominal wall are: skin, superficial fascia, deep fascia,

muscles, transversalis fascia and the peritoneum. The main structure holding the abdominal viscera is the transversalis fascia, which is supported by a group of muscles—the m. rectus abdominis in the central

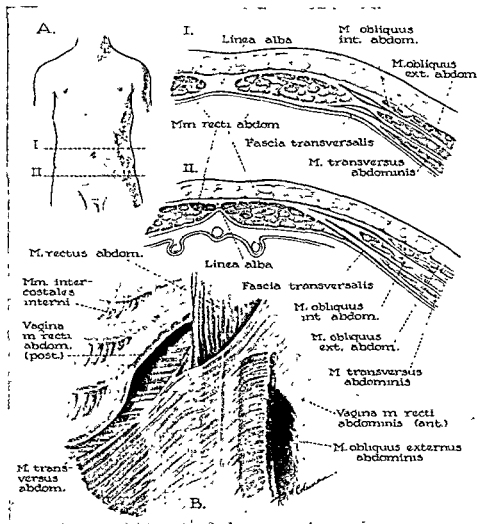


FIG. 34. Formation of sheaths of the rectus muscle.

A. Cross section

I. Above the umbilicus

II. Below the semicircular line.

B. Dissection showing splitting of aponeurosis of the internal oblique muscle into anterior and posterior lamellae (after Spaltcholz).

part, and the external oblique, internal oblique and transversus abdominis muscles in the lateral parts of the abdomen. These lateral muscles terminate as aponeuroses which form the anterior and posterior sheaths of the rectus muscles (the aponeurosis of the external oblique fuses with the anterior lamella of the internal oblique forming the an-

terior sheath and the posterior lamella of internal oblique fuses with the aponeurosis of the transversus abdominis muscle forming the posterior sheath).

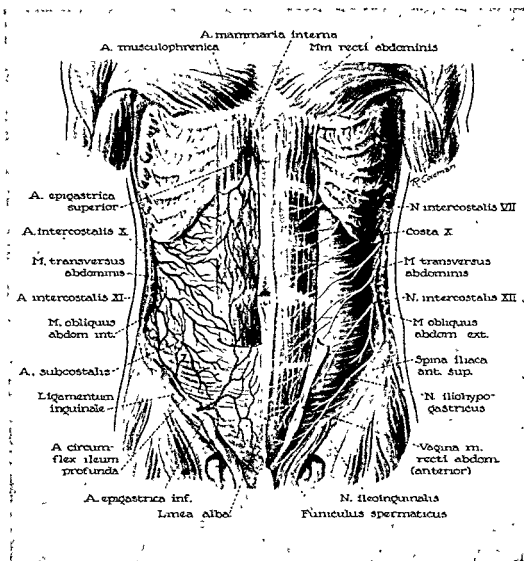


FIG. 35. Nerve and blood supply of the anterior abdominal wall.

While the fibers of the m. rectus run vertically, those of its sheath run horizontally or obliquely. Therefore, if we wish to split the fibers of the rectus muscle longitudinally and are making a longitudinal incision through its anterior sheath we are cutting the fibers of the sheaths transversely, that is perpendicularly to their course.

The lateral muscles of the abdominal wall consist of three layers (external oblique, internal oblique and transversus abdominis), the fibers of which run in an oblique or perpendicular direction to each other. One cannot, therefore, open the abdomen by merely splitting the fibers in the

same direction, but must cut at least one or, in many places, two layers. There is only one way how one can open the abdomen without cutting the muscle fibers, that is by a gridiron incision. However, this incision, even though large through the skin becomes very small when the peritoneum is reached and therefore does not offer a good operative field for most operations.

The blood vessels of the abdominal wall run in two directions: the superior and partly inferior epigastric vessels vertically, the lower intercostals (X, XI, XII) and all lumbar vessels transversely, the superficial and partly the deep epigastric, superficial and deep circumflex iliac obliquely. In other words, no matter in what direction one makes an incision, some damage to some blood vessels will take place. Fortunately, however, the distribution of the blood vessels is such that enough blood supply is left, so that the division of a few branches does not disturb the nutrition of the wall.

The nerve supply of the abdominal wall is derived from the 7th to 12th thoracic nerves and from the ilio-hypogastric and ilio-inguinal branches of the first lumbar nerve. The intercostal branches run horizontally and parallel at a distance of $1\frac{1}{2}$ inches to each other. In making a longitudinal incision of 4 to 6 inches, one is liable to cut 2 or 3 nerves.

If one analyzes the mutual relationship of muscular fibers, blood vessels and nerves one sees that with the exception of a gridiron incision it is impossible to open the abdomen without injuring either the muscles, blood vessels or the nerves. As already mentioned, injury to vascular branches is not dangerous owing to its rich supply. Therefore, the question arises which is less harmful to injure—the nerves or the muscular fibers?

In the early period of abdominal surgery it was considered that muscles should be spared, even if this can be achieved by cutting the nerves. At that time longitudinal incisions were prevalent. P. Assmy, in 1898, demonstrated that muscles cut and resutured were less damaged, than those which were not divided but whose nerve supply was damaged. In other words, he demonstrated that it is a lesser evil to cut the muscle than its nerve supply. This observation, corroborated later by numerous investigators, led surgeons to use preferably transverse incisions in which the nerve supply is preserved. Another advantage of transverse incisions is that through contraction of the lateral muscles, there is no tension on the suture line.

Transverse incisions known as "physiologic incisions" are now used by an increasingly large number of surgeons. Many valuable data are now available as to their usefulness. It was found that by this type of incision

1. Closure of the abdomen is easier because there is less tension of the separated lips of the wound.
2. Patients have less pain while coughing and, therefore, are not

afraid to cough, so that ventilation of the lungs is attained and, therefore, postoperative pneumonia occurs less frequently.

3. Disruption of wounds after operation is considerably less frequent than after longitudinal incisions.
4. Incisional or ventral hernia and postoperative adhesions are considerably rarer after transverse than after longitudinal incisions.

There is described a great number of abdominal incisions which may be divided into five large categories:

- A. Transverse.
- B. Longitudinal.
- C. Combination of the two.
- D. Flap
- E. Gridiron.

The type of incision is not influenced by a consideration whether operation is an emergency, urgent or one planned days or weeks ahead. Whenever at all possible an incision should be made transversely because it is physiologic. For gall-

bladder or biliary tract operations a good exposure is afforded by a Courvoisier or Mikulicz-Kausch incision (Fig. 36); if additional room is needed the latter incision may be enlarged by making a short limb cutting the skin, fascia and splitting the external oblique muscle perpendicularly to the first incision and transforming it into a Sprengel incision (Fig. 36). For gastrectomy a transverse transrectus incision cutting both recti muscles (Fig. 36) affords excellent exposure. For gastrostomy (Fig. 37) we use a short (3-inch) left midrectus longitudinal incision, which though not physiologic offers some advantages for fixation of the stomach and its tube to the abdominal wall, which is not afforded by a transverse incision. Usually the condition for which gastrostomy is done is such that one is not much concerned about the possibility

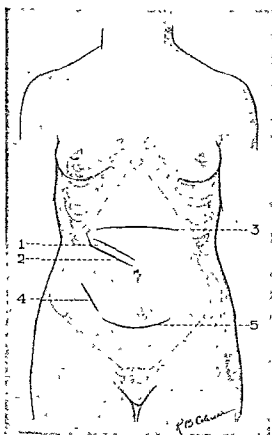


FIG. 36. Transverse and oblique abdominal incisions. 1. Mikulicz-Kausch. 2. Sprengel. 3. Bilateral transrectus incision. 4. McBurney (McArthur) incision. 5. Pfannenstiel incision.

of a future ventral hernia. For gynecologic operations the Pfannenstiel incision (transverse incision of the skin, fascia, anterior sheath of the rectus muscles, separation of the muscles, longitudinal incision of the transversalis fascia and the peritoneum) affords excellent exposure (Fig. 36) and causes less damage than the ordinary infraumbilical midline

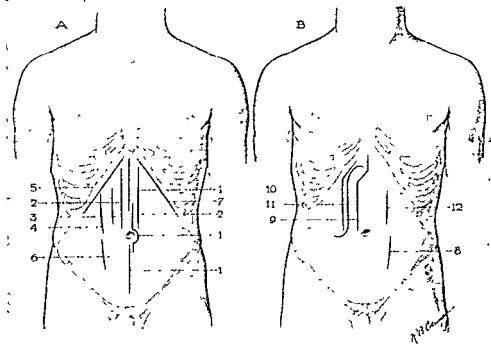


FIG. 37. Longitudinal incisions. A. 1. Supra- and infraumbilical midline incisions. 2. Right and left paramedian incisions. 3. Right midrectus incision (Riedel). 4. Right upper pararectus incision (Lawson Tait, Langenbuch). 5. Kocher incision. 6. Battle incision (Lennander, Jalaguier, Kammerer). 7. Fenger incision. B. 8. Left lower pararectus incision. 9. Kehr incision. 10. Mayo-Robson (Körte) incision. 11. Bevan incision. 12. Left midrectus incision.

incision (Fig. 37). For appendectomy—the McBurney or Battle (Jalaguier) incision is preferable (Fig. 36). Those interested in further details on abdominal incisions are referred to the author's book "The Surgical Technic of Abdominal Operations."

The vaginal, lumbar and subdiaphragmatic approach to the abdominal cavity are discussed in the corresponding chapters on gynecologic, urologic and thoracic surgery. In poor operative risks necessitating operations to be concluded in the shortest possible time, longitudinal incisions should be made, as opening and closure of the abdomen consumes less time than by transverse incisions.

TECHNIC OF OPENING AND CLOSURE BY A TRANSVERSE INCISION. AS

an example of a transverse abdominal incision, we shall describe one for the exposure of the gallbladder.

Step 1. The skin, superficial and deep fascia are divided starting from the lower end of the right costal arch and proceeding mediad to the midline (Fig. 38A).

Step 2. The anterior sheath of the right rectus muscle is cut transversely. It is a good practice *before cutting* the anterior sheath to remove with a piece of gauze the fat which often covers it. If this is not done, one may later find difficulty in visualizing the anterior sheath which is important for the proper closure of the abdomen.

Step 3. Each lip of the divided anterior sheath is sutured to the muscle at three places, (Fig. 38B). It is not necessary to include in the bite the entire thickness of the muscle, but just enough to prevent retraction of the fibers after division of the muscle.

Step 4. The rectus muscle is cut transversely (Fig. 38B).

Step 5. The posterior sheath and the peritoneum are cut transversely (Fig. 38B).

CLOSURE OF ABDOMEN OPENED BY A TRANSVERSE INCISION. The importance of a careful closure of the abdomen can not be over-emphasized. The peritoneum should be properly approximated so that no gaps are left. If small openings are left between the peritoneal lips, tabs of omentum may enter, act as wedges and be responsible for an eventual incisional hernia and postoperative adhesions, complication which must be avoided. The transversalis fascia should be caught in the suture; otherwise even if the peritoneum is carefully sutured, a hernia still may ensue. One has to remember that when one makes an incision, and particularly a longitudinal, the transversalis fascia retracts farther away than peritoneum or the posterior sheath; therefore, if during suturing the posterior sheath the needle catches the lip close to the edge, it may include the peritoneum and the posterior sheath and miss the transversalis fascia, a faulty technic likely to have hernia as a consequence.

Step 1. The peritoneum, with the transversalis fascia and the posterior sheath of the rectus muscle is grasped at four places with artery forceps at the lateral and medial angles and at the middle of the upper and the lower lips. A continuous suture is made from the medial to the lateral angle. We use chromic catgut No. 1 (Fig. 38, C).

Step 2. The upper part of the divided muscle with its anterior sheath is sutured to the lower part by three interrupted vertical mattress-sutures, as follows: The suture enters the anterior sheath of the rectus muscle and passes through the muscle itself at a distance of half of one inch from the cut edge of the lower lip. Then it penetrates the upper lip in the opposite direction passing through the muscles and the anterior sheath and emerging at a distance of half of one inch from the cut edge

of the upper lip. Then the direction of the needle is reversed, and it penetrates the edge of the anterior sheath of the upper and of the lower

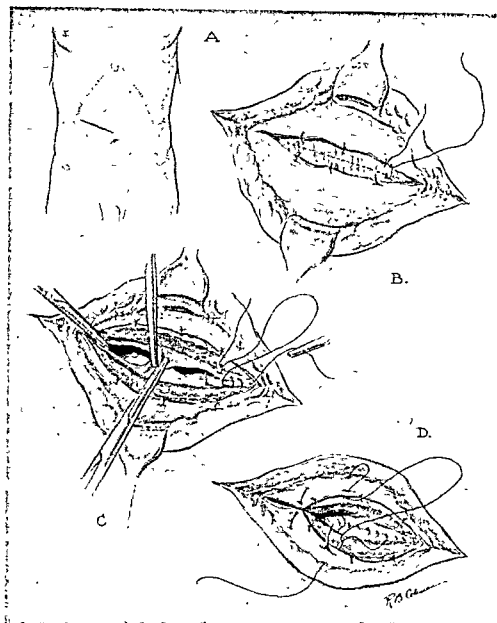


FIG. 38. Opening and closure of the abdomen by a transverse incision. A. Mikulicz-Kausch incision (surface marking). B. Anterior sheath of rectus muscle is sutured to the muscle (dotted line indicates the line along which are cut the muscle and peritoneum). C. Closure of peritoneum, transversalis fascia and the posterior sheath. D. Closure of the muscle by a vertical mattress suture.

lips. In this way a vertical mattress-suture is made. Several such sutures are inserted. A few superficial ones connecting only the anterior sheath are placed between the mattress-sutures (Fig. 38, D).

Step 3. The skin is sutured by any method preferred by the surgeon.
METHOD OF OPENING THE ABDOMEN BY A LONGITUDINAL RIGHT MIDRECTUS INCISION.

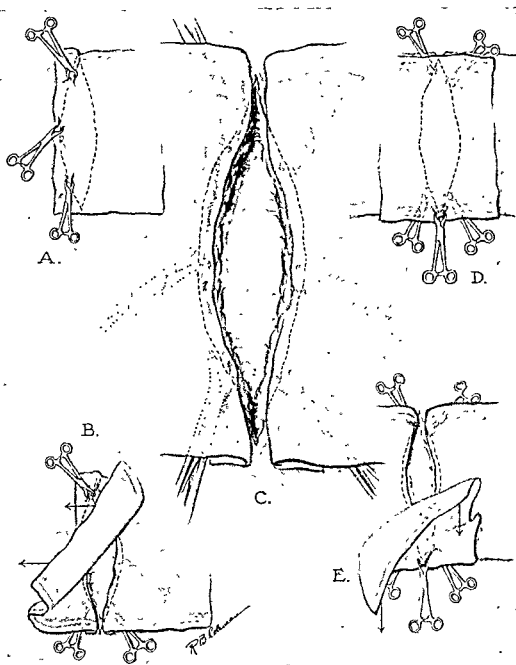


FIG. 39. Opening the abdomen by a longitudinal incision.
 A method of attaching the towels.

Step 1. The skin and superficial fascia are cut for a distance of four to six inches.

Step 2. Towels are clipped to the lips of the wound; they are held

with three towel forceps to each lip—one at the upper, the other in the middle and the third at lower end of each lip. The towel forceps are placed so that the towels cover them (Fig. 39).

Step 3. The fat is cleansed from the underlying anterior sheath for a width at least of three-fourths of an inch from the intended line of

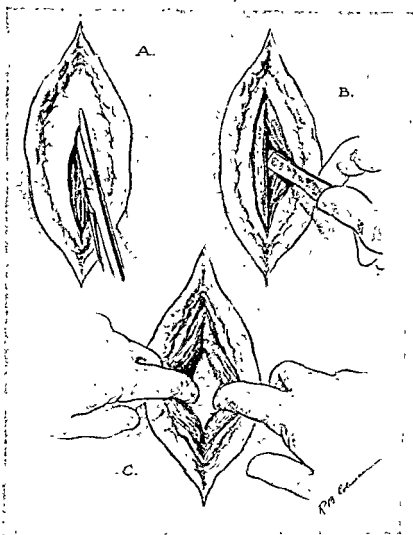


FIG. 40. Opening the abdomen by a longitudinal right midrectus incision. A. Anterior sheath incised (down with scalpel, upward with scissors). B. Muscle split with handle of scalpel. C. Muscle further split with index fingers of both hands.

incision through this sheath. This greatly facilitates its future closure; if this is not done, the anterior sheath is covered with fat and one is liable to catch fat instead of the anterior sheath in closing the abdomen. After the anterior sheath is incised (40, A) the muscle is split with the handle of the knife (Fig. 40, B) either through the entire length of the

incision or sufficient to introduce the index fingers of both hands, which then split the muscle fibers to the desired length (Fig. 40, C).

Step 4. The posterior sheath (with the transversalis fascia and peritoneum) is now grasped by two toothed tissue forceps at a distance of one-fourth of an inch from each other. With the thumb and index finger

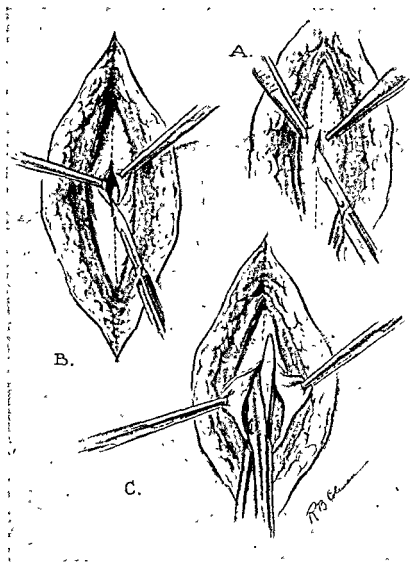


FIG. 41. Opening the abdomen by longitudinal right midrectus incision. A. The post. sheath, transv. fascia and peritoneum are nicked with a scalpel. B. Incision being enlarged downward with a scalpel. C. Incision enlarged upward with scissors.

the surgeon feels whether any viscus is adherent to the peritoneum between the forceps. If there are no adhesions, the fold is nicked open with a knife. The edges of the opening are then caught by artery forceps at two places, one on the surgeon's and the other on the assistant's side. The opening is now enlarged by cutting with scissors upward and with the knife downward (Fig. 41).

CLOSURE OF ABDOMEN OPENED BY A LONGITUDINAL INCISION. We suture the abdominal wall in four layers.

1. The first layer is composed of peritoneum with the transversalis fascia and posterior sheath. It is closed with chromic catgut No. 1. We usually employ a continuous suture, though occasionally we resort to interrupted sutures. In the latter case the distance between two sutures is about one third of one inch. It is extremely important to penetrate this layer not at the edges but at least one-fourth of an inch away so as to be sure that the transversalis fascia is included, as it retracts and if the bite is taken at the edge it may not include it. Omission of the transversalis fascia in the bite is probably the most frequent cause of incisional hernia.

2. The second layer consists of muscles, which we close interruptedly with plain catgut No. 1. The distance between two bites is one inch.

3. Third layer (the anterior sheath) is closed with chromic No. 2 single or No. 1 double thread by continuous suture. If interrupted sutures are made the distance between two bites is about one-third of one inch.

4. The fourth layer includes the skin. We use horse-hair or dermal and close it by a continuous vertical mattress-suture. In stout patients, or when large longitudinal incisions are made "tension sutures" of silk-wormgut are useful. They penetrate the entire thickness of the abdominal wall except the peritoneum and are placed immediately after the peritoneum is sutured. They are tied after the skin is closed over a strip of gauze.

CLOSURE OF THE ABDOMEN IN DIFFICULT CASES. In some cases one may have difficulty in closing the abdomen. This is particularly so with obese patients, or when patients do not take the anesthetic well and are not completely relaxed. Under such conditions the peritoneum may tear, which proves to be the main cause of postoperative adhesions and in some cases of postoperative hernias.

This can be overcome in several ways:

- a. Insertion of sutures through the entire thickness of the abdominal wall except the skin by a series of interrupted sutures. These sutures are placed half of one inch apart and tied only after all of them have been inserted.
- b. Insertion of several sutures through the entire thickness of the abdominal wall except the skin. These sutures are placed at a distance of one and one half inches from each other and the ends are not tied, but clamped by hemostats (Fig. 39, A). Next, the peritoneum is caught by hemostats at several places, including one at the upper and the other at the lower angle of the incision. A suture is inserted at the upper peritoneal angle and the ends are tied. Then, the same suture is inserted as a continuous over-and-over suture to close the upper half of the peritoneum in a loose manner so as

not to tear the structure (Fig. 42, A). Next, the tension suture at the upper angle is rendered taut so as to bring the lips together. The thread inserted loosely in the peritoneum is now pulled in the direction of the suture line, thereby closing the peritoneum without producing any tears (Fig. 42, B). The finger is then introduced

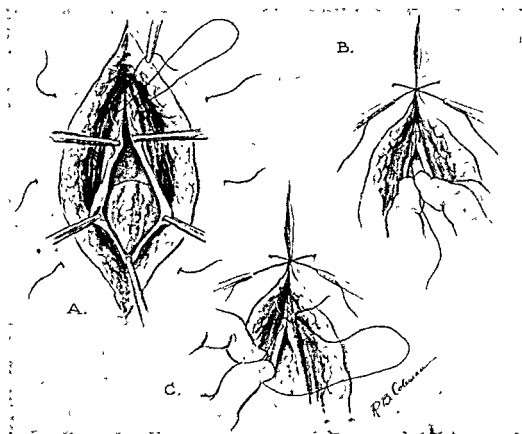


FIG. 42. Closure of abdomen in difficult cases. A. Peritoneum grasped with hemostats, traction sutures in place, loose continuous suture in upper portion of wound. B. First traction suture drawn tight, peritoneal suture drawn taut in direction of line of suture. C. "Knotless knot" made in peritoneal suture by means of back stitch.

into the peritoneal cavity through the lower half of the incision to make sure that no omentum or portion of bowel wall is caught between the peritoneal lips (Fig. 43, D). A back stitch is carried through the peritoneum immediately back of its closed portion. This stitch acts as a "knotless knot," thus preventing the peritoneum from giving away; it is placed under control of the finger in the peritoneal cavity to make sure that no loop of bowel was caught with the needle. The lower half of the peritoneum is sutured in the same loose manner as with the upper one; the tension suture in this part is placed on stretch until the edges of the lips come into approximation. The loose thread inserted in the peri-

toneum is now pulled in the direction of the incision and the lower portion of the peritoneum is now closed. The anterior sheath is then closed either by a series of interrupted sutures or by a continuous one (Fig. 43, E). The ends of the traction sutures are now tied.

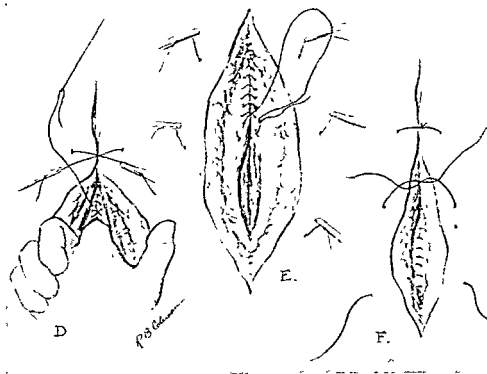


FIG. 43. Closure of abdomen in difficult cases. D. Examination of peritoneal cavity with index finger to make sure no omentum, bowel, etc., are caught in the suture. E. Continuous suture of anterior rectus sheath. F. Traction sutures tied.

Complications

Among the different complications arising after laparotomy, the following are important:

- a. disruption of the wound;
- b. incisional hernia;
- c. infection of the abdominal wall and cavity;
- d. abdominal adhesions;
- e. foreign bodies left in the abdomen; and
- f. keloids and ossification of the scar.

A. DISRUPTION OF THE WOUND. This is separation of the lips of the abdominal wall before firm union is established. It may be accompanied by protrusion of omentum or intestine outside the abdominal cavity,

when one uses the term *evisceration*. This complication occurs in about 1.5 per cent of all laparotomies. It usually occurs between the 5th and the 9th postoperative day, but may take place as early as the first and as late as the sixtieth day. Age, sex or race play no rôle in its development. It occurs more frequently with debilitated, anemic patients or individuals with a fat abdominal wall. Any disease which retards the healing of wounds may predispose to this particular complication. Patients with insufficient vitamin C values, with hypoproteinemia and catgut allergy are more often affected than other types of individuals.

It is a well observed fact that this condition occurs more frequently after longitudinal than transverse incisions, the suture line in the former being under direct pull in the opposite direction whenever there is a contraction of the lateral abdominal muscles. However, as already mentioned in describing the technic of closure of longitudinal incisions, we believe that another important contributing factor is retraction of the transversalis fascia beyond the lip of the peritoneum and the posterior sheath of the rectus muscle which, therefore, often is not included in the suture.

With exception when a patient is allergic to catgut, it is immaterial what kind of suture material is used. Disruption of wounds occurred in cases when absorbable as well as non-absorbable suture was used. With patients allergic to catgut disruption may occur within a few days after operation when no traces of catgut are found. Disruption occurs irrespective whether the wall is closed by through-and-through sutures or in layers and whether "tension sutures" are used or not.

Drainage of the abdominal cavity favors disruption. Statistics show that it occurs considerably more often in cases in which the abdomen was drained. For this reason it is advisable to carry the drain through a stab wound and close the original incision firmly.

Infection undoubtedly predisposes to disruption since it delays healing of the wound.

Increased intra-abdominal pressure by coughing, vomiting and gas distention are contributing factors.

Symptomatology. The symptoms vary depending on whether there is only disruption or also evisceration with or without concomitant strangulation of the bowel. Often the patient feels fairly comfortable and states that "something gave away" while he coughed or vomited. Pain and shock are rather uncommon signs. Vomiting is occasionally present. On examining the disrupted wound one finds a sero-sanguinous discharge in about 20 per cent. Any edema or fluctuation along the line of skin incision should arouse suspicion of the possibility of disruption of the deeper structures of the wound.

Treatment. There is no unanimity regarding the treatment of a disrupted wound. Three measures are open to the surgeon:

1. **Bringing the edges of the disrupted wound together with adhesive plaster.** Before doing this the patient gets a dose of morphine; the skin around the wound is washed with soap and water; a sterile gauze is placed over the disrupted wound. The edges of the wound are then brought over the gauze and kept together by wide strips of adhesive plaster. If there is evisceration the patient must be subjected to infiltration analgesia around the lips of the wound or spinal analgesia under which the viscera are replaced, sterile gauze is placed over and adhesive tape brought over it. This gauze is removed gradually.

2. **Secondary suture of the abdominal wall.** As soon as the diagnosis is made a dose of morphine is administered and the patient taken to the operating room. The abdominal wall is cleansed before infiltration analgesia is carried out. If, however, inhalation anesthesia is preferred, the wall is cleansed after its induction. Closure may be accomplished either by through-and-through silkwormgut suture or in layers. In the latter case silk, silkwormgut or steel wire sutures may be employed.

3. **Approximation of the edges with adhesive plaster over a tampon followed in a few days by secondary suture.**

We believe that if the patient is not in shock it is better to start with the secondary suture at once because it reduces the convalescence from five days to two and a half weeks, and is less liable to be followed by post-operative adhesions.

Mortality in either method of treatment is about 35 per cent. This depends less on the method of treatment than the general condition of the patient.

B. INCISIONAL HERNIA. The symptomatology and the treatment are discussed in the chapter on hernia.

C. INFECTION OF THE ABDOMINAL WALL. In varying degrees it is quite common after laparotomy. In most of the cases it is so slight that the wounds are considered as clean; in others the infection is more extensive, but still does not extend in depth and appears as stitch supuration or small abscess formation in the subcutaneous tissue. The microorganisms found in such cases are staphylococci, streptococci and *B. coli*.

The treatment is generally that of infected wounds, namely prevention of the spread of contamination by strictly aseptic changes of dressings, adequate drainage of accumulated pus, Dakins solution for irrigation and, lastly, the application of sulfa drugs in powdered form or in solution. However, in some cases considerable destruction of tissues takes place. They become invaded by anaërobes and treatment then becomes a difficult surgical problem. Among these conditions two should be kept in mind, namely, postoperative progressive gangrene of the skin and gas gangrene infections.

Postoperative progressive gangrene of the skin (Progressive bacterial

synergistic gangrene). This disease is very rare. Up to now the number of cases described is about 50. It usually affects the skin and subcutaneous tissue only and does not reach deeper structures. It originates either from the wound edges, or from the suture canals, though occasionally it may develop in the vicinity of the wound without being directly connected with it (Lichtenstein); it usually develops after laparotomy, but in few cases it involved the chest wall. In most of the instances it developed after a drain had been employed, but in some cases the abdomen was closed without drainage. The first case of this kind was reported by Luckett in 1909. The clinical picture is quite typical. It starts two days to four weeks after the primary operation with patches of the reddened skin appearing around the wound. This process starts either from the vicinity of the drain or the skin suture channels. After a few days the skin attains a bluish tint and later becomes dark blue and still later when gangrene is fully developed the color is black. When the picture is fully developed, one can differentiate three typical zones of changes in the skin; the central portion is gangrenous, the more peripherally situated is red blue, swelled and a few centimeters wide; the most peripherally situated is markedly red and fuses with the normal skin. Characteristic for this type is that it affects only the skin and superficial fascia and not the deeper structures. Another characteristic feature is the extreme tenderness of this area. The pain while dressing the wound is so severe that in some cases general anesthesia becomes necessary. This condition, if treated conservatively with antiseptic solutions, vaccines, sera and the like, invariably progresses more or less rapidly. In some cases it may affect the entire abdominal wall and spread to the chest wall. In the beginning it does not affect the general condition, later, however, the patient becomes toxic. The temperature is either normal or subfebrile.

While gangrene of the skin may be produced by streptococci, amebae, diphtheroid bacilli and staphylococci, this particular type is produced by a variety of streptococcus for which is coined the term "*micro-aërophilic streptococcus*" in association with staphylococcus. Meleney considers that the presence of both of these varieties in the same wound will produce this particular type of gangrene, while either the micro-aërophilic streptococcus or staphylococcus alone may also produce a gangrene of the skin, but not this particular type. He called this type "*postoperative progressive bacterial synergistic gangrene.*"

Though medical treatment with antiseptic solutions or ointments leads occasionally to a cure, the best method is radical excision of the skin of the entire area, cutting the healthy tissues for a distance of at least one inch from the diseased area. After that the wound is treated according to established principles of treatment of wounds. Meleney advocates the use of an aqueous solution of zinc peroxide in the form

of a paste not only for this particular type of gangrene but for any infection produced by anaerobes; he believes that freed oxygen will check the further growth of the anaerobes.

Gas gangrene infection of the abdominal wall is a very infrequent complication, to date only about 60 cases having been recorded. It occurs usually after operations on the large bowel, particularly for suppurative appendicitis and intestinal obstruction and considerably rarer after operations on the stomach or duodenum. It starts usually 36 to 72 hours after surgical intervention by moderate elevation of the temperature and a rapidly increasing pulse rate entirely out of proportion to the temperature. Thus, while the temperature ranges between 100 and 101°F. and later drops to normal or even subnormal level, the pulse beats range between 120 to 140 and become thready. The patient is restless; the respiratory rate is increased; there is dyspnea and air hunger. The face and mucous membranes are pale and the patient lies in deep prostration. This paleness is due to destruction of the red blood cells by anaerobic hemotoxins.

Jaundice is quite frequent due to hepatotoxic action of the anaerobes. Examination of the wound reveals the skin to be at first red and markedly swelled. Pressure on the wound produces a sero-sanguinous exudate escaping from the suture line together with gas bubbles. The mortality rate is about 60 per cent. Death occurs usually a few days after the onset, though in some cases it took place as early as 14 hours after the onset.

The diagnosis is made bacteriologically. There are many methods described, some of them facilitating the discovery of the anaerobes within 10 to 12 hours, thus enabling the surgeon to start immediate treatment. This condition should be differentiated from erysipelas, septic cellulitis and septicemia.

Treatment. The best method of treatment is a combination of surgery, antitoxin injection and chemotherapy with sulfa drugs. Surgery consists of opening of the wound, removing necrotized tissues, inserting drains to provide escape for the necrotized tissues and exudates. Then, the wound is dusted with 5 gm. of sulfanilamide, sulfathiazole or sulfapyridine powder. The patient also is given sulfanilamide, sulfathiazole or sulfadiazine orally—2 gm. as the first dose and then 1 gm.—three times daily until the condition improves. Several reports of beneficial action of penicillin in treatment of gas gangrene recently have been published.

Antitoxin treatment consists of intravenous injection of 7500 units of clostridium welchii antitoxin, 3750 units of clostridium septicum and 2500 units of clostridium oedematiens. These doses are repeated as often as necessary, as long as symptoms of intoxication persist. There are several favorable reports of combating the gas bacillus infection by deep x-ray therapy and also by the local application of zinc peroxide.

D. ABDOMINAL ADHESIONS. This is quite a frequent sequela of any surgical procedure. Though in many cases it yields hardly any symptoms, in other cases it gives rise to pain and in some even intestinal obstruction—incomplete or complete. Statistical study of a large number of cases shows that in nearly 75 per cent of all cases of intestinal obstruction there is a history of a previous abdominal operation.

This is the reason why many attempts were made to determine the causes of the formation of adhesions and their prevention.

Among the theories propounded one of the most generally accepted is **inflammation**. According to this theory, an inflammatory process, which originally starts at some place in the mucosa of the gastro-intestinal tract travels through the muscularis and serosa, then spreads through the lymphatics to the neighboring parts of the abdominal cavity and even to the distant points from the starting place and produces exudates, which become organized, thus forming adhesions. In this way adhesions are formed even if no operation was performed.

According to followers of this theory, contamination of peritoneum takes place during operation with ensuing formation of adhesions.

However, there are many observations which cannot be explained by this theory. Many times operations were done for interval appendicitis in which no adhesions were found at the time of operation, but were found to be extensive at some future time when patient was operated for some other condition. On the other hand, not infrequently patients who have been operated for a perforated stomach, gallbladder or appendix, in which contamination took place, undergoing much later another abdominal operation do not show the presence of any adhesions.

Other surgeons believe that the formation of adhesions is attributable to a constitutional predisposition. Payr and Vogel many years ago stated that the asthenic type of individual is more liable to form adhesions than stout one.

The majority believe that adhesions are most liable to be produced when the endothelium of the viscera or of the peritoneum is injured. This injury usually takes place by rough handling and by rubbing with dry gauze. Therefore, rough handling should be avoided and all raw surfaces should be carefully peritonized. Another source of injury is irritation of the visceral peritoneum by the antiseptic solutions used for the preparatory disinfection of the skin. This material may come in contact with viscera brought outside the abdominal cavity and kept on the abdominal wall. Therefore, all viscera delivered outside the abdominal cavity should never be allowed to come in contact with skin.

With all these considerations, there are cases when in spite of all precautions adhesions are formed requiring repeated laparotomy for their separation. Fleisch-Thebesius reported a case in which the patient was operated 23 times for adhesions.

The treatment is essentially prophylactic, even though there is no single

method which prevents the formation of adhesions. One has to handle the tissues very gently, never rub them with gauze nor bring them in contact with the skin. Careful control of bleeding is essential. The edges of the peritoneal lips should be in close approximation so as to leave no gaps.

No drains should be inserted unless there is an absolute necessity. Raw surfaces should be peritonized. All these measures serve as preventive.

Among other attempts to prevent the formation of adhesions should be mentioned of covering traumatized surfaces with camphor oil, olive oil, vaseline, amniotic or vitreous fluid of the eye, and the like. However, once adhesions are formed little can be done for their dissolution. Different solutions were tried to digest them, such as pepsin, tripsin and papain. While some authors reported good results with papain (in 1:50.000 concentration) others experienced failures. Others tried to prevent adhesions by promoting peristalsis of the bowel, by changing the position of patients in bed and later by massage of the abdomen, but the mere existence of so many remedies shows that results obtained by them so far have proved ineffective.

E. FOREIGN BODIES LEFT IN ABDOMEN. Though cases are known of foreign bodies left in the pleural cavity, in the pericardium and in the neck behind the sternohyoid or sternothyroid muscles after thyroidec-tomy, for evident reasons, most frequently they are left in the abdominal cavity and particularly in gynecologic operations.

As a matter of fact, the first such case was recorded in 1859 by Ols-hausen, a great gynecologist in those days. It is estimated that foreign bodies are left four times as frequently in pelvic than in other abdominal operations. It is difficult to say how often a foreign body is left in the abdomen. Many times such cases are not reported when they occur; in others the forgotten foreign body may not give symptoms and, therefore, never discovered. However, that such accidents do occur even now in spite of all precautions, is a matter of record. It is usually considered that in one out of 1000 laparotomies a foreign body is forgotten in the abdomen. Small stick sponges, less frequently large laparotomy sponges, hemostats, hooks, glass rods, towel clips and pins are among the forgotten objects.

In about 10 to 15 per cent, the foreign bodies cause immediate complications, most frequently pain and peritonitis, so that the patients have to be reoperated before they have a chance to leave the hospital. In this group the mortality is high, reaching about 50 per cent.

In the majority of cases the foreign bodies become encapsulated or occasionally remain silent in the sense of causing no symptoms. The longest case on record is Ossipov's in which the foreign body was encapsulated in the abdomen for 25 years. Though this group of encapsu-

lated foreign bodies does not give such an high mortality as the previous group it still reaches to about 25 per cent. There were offered many suggestions how to obviate or even to make impossible the occurrence of this accident. However, none has proved fool proof. Large laparotomy sponges should have a tape, clamped by an artery forceps which should be kept outside the abdominal cavity. Irrespectively whether they do have or do not have tape, it is inadvisable to permit the entire length of the laparotomy sponge to be inserted within the abdominal cavity; one end of it always should be outside the abdomen. It is much easier to lose a stick sponge, for which reason they should not be left for hemostatic purposes, but used only for sponging and immediately taken out. It certainly is not advisable to have several stick sponges at the same time, especially for the control of bleeding.

However, the surest way of not forgetting any object is care on the part of the surgeon and his first assistant. They should always think of such a possibility and that it has happened even to the greatest masters in surgery. It goes without saying, that immediately before the closure of the peritoneum all the sponges and instruments should be counted.

F. KELOIDS AND OSSIFICATION OF THE SCARS. Keloid transformation of the scar is not an infrequent occurrence. It occurs often in negroes. Apart from being unsightly, it does not give any trouble. Ossification of a scar with actual bone formation is quite a rare occurrence, 70 such cases having been reported to date.

Drainage of the Abdominal Cavity

Drainage of the abdominal cavity in frank peritonitis or potential peritonitis after laparotomy was an accepted surgical measure in the last century. Mikulicz stressed the folly of routine drainage of the abdominal cavity as early as in 1881 and urged to do it only in definite conditions. Yates, in 1904, showed on dogs that a drain introduced into the abdominal cavity through the abdominal wall fails to communicate with the general peritoneal cavity six hours after its insertion. Carmine injected into the cavity through another opening did not escape through the drain tract, even if a large amount and under increased pressure was introduced. In other words, the drain was walled off by adhesions from the rest of the peritoneal cavity so completely that even fluid could not penetrate this barrier. He also demonstrated that in the presence of peritoneal infection this six hour period was shortened and the adhesions which formed were more dense and harbored micro-organisms for a longer period than tracts formed in the abdomen that were not previously infected.

F. Rost showed that fluid injected through a drain inserted twelve hours previously could not reach the general peritoneal cavity but was regurgitated through the space between the drain and the abdominal lips.

These experimental findings were in accord with clinical observations made by several surgeons at about the same time. Hotchkiss, in 1906, reported a group of 28 cases of acute appendicitis with ruptured appendices of which 16 were not drained with only one lethal case and 12 were drained with four deaths. Similar results were recorded by Bauer in a study of 88 cases published in 1911.

P. Clairmont and M. Meyer, in 1926, reported 176 cases of perforated appendix of which 152 had the abdomen closed without drainage. Their conclusion was that though secondary abscesses in the abdominal cavity were formed more often in cases which were not drained, a larger number of patients had a smooth recovery and remained permanently in good health under non-drainage than those whose abdominal cavity was drained.

Other surgeons had the same experience. Marchini found that in cases of ruptured appendices with local peritonitis, those without drainage gave a 1.7 per cent mortality as compared with 5 per cent for drained cases. Patients with diffuse peritonitis who were not drained gave 20.6 per cent mortality as compared with 30.6 per cent for those in which drainage was instituted.

The reasons why drainage increases the mortality and morbidity rate may be summarized as follows:

1. A drain in a few hours ceases to drain; it becomes a foreign body walled off by adhesions from the general peritoneal cavity.

2. The drain increases formation of adhesions with potentially partial or complete intestinal obstruction and or fecal fistula, activating an already existing peritonitis.

3. The drain irritates the abdominal wall and cavity producing purulent discharge, which, by gravity, reaches and remains at the bottom of the wound and becomes an excellent culture medium for pathogenic micro-organisms. Some of this infected fluid overflows the channel and escapes through the drain; this was wrongly interpreted by many as proof that the drain does the work by diverting outside the purulent contents of the peritoneal cavity.

With the advent of chemotherapy the use of drains became still more limited and *it is now relegated only to cases with a circumscribed abscess*. In all other cases of local or general peritonitis, the abdomen is closed and 5 gms. of sulfanilamide or sulfathiazole is put into the peritoneal cavity before closure of the abdomen and followed by the parenteral or oral administration of sulfa drugs and of penicillin.

EARLY RISING. Early rising after laparotomy is by no means of recent origin. Emil Ries, of Chicago, was probably the first who used to employ it since 1899.

However, it passed nearly unnoticed until Krönig advocated it in Germany, in 1906, and it was only in the late twenties that it was advo-

cated in some clinics. Even now it is used only by a comparatively small number of surgeons. However, the experience with this method runs into many thousand of cases and, therefore, some definite conclusions as to its value can be reached.

Early rising can be classed as:

- a) **Immediate rising**, when the patient leaves the operating table and walks, assisted or unassisted, to his bed; and
- b) **Early rising** when the patient gets up from the bed on the third postoperative day and is up and around.

The reason why some surgeons advocate early rising is because it lessens such complications as retention of urine, obstipation, colicky pain, pulmonary embolism, and thrombophlebitis. Any fear of eventration is not justified as shown by the collected statistics. Thus, Manuel Giagni reported from Hospital de Asunción of Rosario, Argentine, 184 cases of immediate or early rising in which no such complications occurred.

His cases are grouped as:

Chronic appendicitis	121
Acute appendicitis	20
Inguinal hernia	21
Hysterectomy	11
Gastro-enterostomy	5
Ovarian cyst	3
Epigastric hernia	2
Ruptured ectopic pregnancy	1
	<hr/>
	184 cases.

Livius Campeanu and M. Papp collected 3197 case reports comprising appendectomies, herniotomies, gastro-enterostomies, gastric resections, cholecystectomies, hysterectomies and extrauterine pregnancies in which the patients either left the operating table immediately or left the bed not later than on the third postoperative day. In all these cases the post-operative complications occurred less frequently than in comparable number of cases in which the patient stayed in bed eight to ten days.

It has to be borne in mind, however, that not all cases can be routinely permitted to rise immediately or early.

After spinal analgesia the patient should stay in bed at least 24 hours even if the operation performed was a simple uncomplicated appendectomy. In case of acute infectious surgical diseases early rising may produce embolism and, therefore, is contraindicated. Among other contraindications to early rising should be mentioned severe myocardial damage and drains in the abdominal cavity.

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Chapter VI

Urgent Surgery of the Stomach and Duodenum

By JULIUS L. SPIVACK

ACUTE PERFORATION OF PEPTIC ULCER

Perforation of gastric or duodenal ulcer is one of the most serious emergencies confronting the surgeon. The sudden onset, acute course, and high mortality place these cases among the most trying experiences.

HISTORICAL NOTE. Perforation of gastric ulcer has been known to the medical profession since the middle of the eighteenth century. Sir John Finch¹ gave an account of the death of Henrietta Anne of England, daughter of Charles I. He reported that the autopsy revealed a perforation in the middle of the anterior wall of the stomach; a large amount of fluid was found in the free peritoneal cavity.

Perforation of a duodenal ulcer was reported in 1746 by Hamberger² of Jena. Later, several reports were published in different countries. One of the most interesting cases was that described by Penada¹ of Padua. It concerned a perforated duodenal ulcer of a butcher thirty five years of age. Penada recorded the symptoms preceding perforation, immediately following it, and the late signs of generalized peritonitis. His description may well be presented by a contemporary surgeon for its lucid accuracy despite its dating back to 1791. This clinical report was succeeded by one of an autopsy finding: The investigator observed about three inches below the pylorus an oblong opening resembling an incision made with a knife, measuring 8 by 2 lines.* The external edge of this cleft was of considerable thickness; to the touch it was hard and indurated. The peritoneal cavity contained a large amount of fluid and undigested food.

Brinton,³ in 1856, gave an excellent presentation on the subject of gastric ulcer.

In the United States the first report on perforation of duodenal ulcer was presented by O'Hara⁴ in 1876 before the Philadelphia Pathologic Society.

Mikulicz,⁵ in 1880, operated upon a patient with a gastric perforation. The patient died a few hours after closure of the perforation. In 1885 and 1887, Czerny performed an unsuccessful operation by suturing a perforated ulcer. Both Van Wahl, in 1889, and Mouisset, in 1890, published the report of a fatal case. Heussner,⁶ in 1892, performed the first successful gastrorrhaphy for perforated gastric ulcer. R. F. Weir,⁷ was probably the first in the United States successfully to perform in 1896 gastrorrhaphy for a perforated gastric ulcer. In all these cases the method consisted of simple closure of the opening, although in some it was difficult to close the gap. The edges were indurated and the sutures cut through so that the opening was enlarged rather than repaired. H. Braun⁸ likewise failed to close the opening at first. In an attempt to over-

* A line is 2.2 millimeters.

come this (1893) he covered the opening with a detached piece of omentum which he fixed to the anterior wall of the stomach by a series of interrupted sutures. Bennett,⁹ of London, in 1896, did not cover the cavity with omentum but used non-detached omentum to plug a gastric perforation; the patient recovered.

In some cases, after closure of an opening, especially close to the pyloroduodenal junction, a stricture of the pylorus ensued. In order to overcome this complication, Braun proposed to supplement gastrorrhaphy by gastrojejunostomy. Haberer,¹¹ in 1919, advocated gastrectomy as an operation of choice. Dowden,¹² in 1909, described his method of treating a perforated ulcer. He excised the ulcer and supplemented this procedure by pyloroplasty.

ETIOLOGY. The literature reveals that cases of gastric and duodenal ulcer have been increasing in number. This applies also to cases of perforation but at an even greater ratio. The actual proportion of ulcerations and perforations is difficult to determine. In some series the ratio is as low as 1 per cent, while in others it reaches 30 per cent. It would seem that the ratio is determined by the type of patients who comprise the series of cases. Observations in private clinics are chiefly made on well-to-do patients who have the means of receiving the best care; therefore, the incidence of perforations is lower than in a group of patients treated in some charitable institution of a large metropolitan area. To arrive at a correct conclusion, therefore, it is necessary to collect statistics of thousands of cases. Bager¹³ found that of 9,474 cases of gastroduodenal peptic ulcer, perforation occurred in 18.1 per cent. Eliason and Ebeling¹⁴ reported an incidence of perforation slightly above 10 per cent in a series of 729 cases of peptic ulcer from the combined records of the University of Pennsylvania and Philadelphia General Hospital. DeBakey¹⁵ found an incidence of perforation of 13.2 per cent in a series of 33,439 cases of gastroduodenal ulceration.

SEX INCIDENCE. Perforation occurs overwhelmingly in the male at a ratio of about 18 to 1. This cannot be explained by the fact that ulcer occurs more frequently in the male and, therefore, perforation is predominant in the male, because the ratio of non-perforated ulcer is only 3 to 1. Probably one reason for this may be the fact that men who have ulcers are more careless in their habits, i.e., excessive physical exertion, intake of large quantities of food, and the like, all of which may precipitate perforation. However, it is interesting to find from reports dating back to the middle of the last century that there was a considerable preponderance of perforations of women over men. At the beginning of the twentieth century the proportion was about equal, and only during the third decade of this century has there been a male preponderance. At the present time many clinics report that about 90 to 95 per cent of all ruptured ulcers occur in male patients. Brinton³, in 1856, stated that 68 per cent of all patients with perforated gastric or duodenal ulcer were women. Judine,¹⁶ in 1939, showed in a series of 1,355 cases that only 2

per cent were female patients. H. L. Thompson,¹² in a series of 500 cases of perforated ulcer, published in 1937, reports 94.2 per cent in male and 5.8 per cent in the female sex. DeBakey,¹³ in a large series comprising about 15,000 cases found that the occurrence in women was only 7.7 per cent. Several surgeons have pointed out that in women perforation of the stomach occurs more frequently than perforation of the duodenum.

AGE. As peptic ulcers occur preponderantly between the ages of twenty and fifty, the same holds true about perforations. Approximately 70 per cent of all cases of perforation take place in this period. Nevertheless, perforations have been recorded in infants two days old, and at the ages between eighty and ninety. Many surgeons have pointed out that perforated duodenal ulcer occurs more frequently in persons under forty, while in those over fifty gastric rupture is more frequent than duodenal perforation.

RACE. With the exception of the Indians all races show about equal proportions in the incidence of perforation. The Indians manifest a ratio five times greater than that of Caucasians, Negroes or Mongolians.

SEASONAL VARIATIONS. There is a slight preponderance of peptic ulcer in the spring and fall over the summer and winter; to some extent this applies also to perforation of these ulcers. While perforation occurs more frequently in the spring and fall, the condition does not differ appreciably from that during the summer and winter.

OCCUPATIONAL VARIATIONS. It is a common belief among physicians that the highly strung individual, especially of the intellectual type, is more frequently subject to perforated gastric and, particularly, duodenal ulcer than others; however, the statistical review of many series does not substantiate this contention.

EXCITING CAUSES of perforation are: excessive consumption of alcohol, physical exertion, foci of infection and trauma of the abdominal wall. Several cases of rupture of a gastric or duodenal ulcer have been reported after fluoroscopic examination.

PATHOLOGY. Peptic ulcers commonly occur as single and round lesions, ranging in size from a few millimeters to 1 cm. in diameter. They may be soft and superficial, when they are acute and of recent origin, or indurated of callous consistency, when they are of the chronic variety. Most perforated ulcers are located close to the gastroduodenal junction. Prior to perforation this area is frequently inflamed and its topography becomes distorted. In many cases it is, therefore, difficult to determine at the time of operation whether the perforation is on the gastric or duodenal site of Mayo's vein. In other words, it is difficult to determine from a gross inspection whether it is a perforated gastric or duodenal ulcer. This was the chief reason why in the past opinions varied widely as to the relative frequency and site of gastric and duodenal ulcers. This is reflected in the statistics of Bager¹³ who claims that from 1,488 cases

of perforated ulcer 30.7 per cent were duodenal, as compared with Judine's¹⁶ report who found in a series of 928 cases 87.5 per cent duodenal ulcer. Judine's claims are based upon microscopic studies of resected tissue and, therefore, his estimates are more reliable. A statistical review of 729 cases by Eliason and Ebeling¹⁷ shows that 80 per cent of all perforated ulcers are on the duodenal site. Generally, it may be stated that most statistical surveys of recent years reveal a preponderance of perforated duodenal over gastric ulcers in an approximate proportion of 80 to 20.

SITE OF PERFORATION. Duodenal perforation occurs in the first portion in about 98 per cent, while perforation in the second portion of the duodenum takes place only in 2 per cent of all cases. In the first portion it occurs about ten times as frequently on the anterior than the posterior wall. Perforation in the second portion occurs three times more frequently on the anterior than the posterior wall.

Gastric perforation occurs on the lesser curvature close to the pylorus in about 70 per cent, and on the lesser curvature close to the cardia in only 15 per cent; perforation in the remaining cases appears on the posterior wall or close to the greater curvature. Generally, the ulcer perforates into the free peritoneal cavity but, occasionally, into some viscus, such as the gallbladder, extrahepatic bile duct; lungs, pancreas, or spleen.

Re-perforations occur in less than 1 per cent of all perforations. Pearse,¹⁸ in reviewing the literature, found 33 re-perforations in 4,183 cases.

Multiple perforations are very rare. Judine claims that in 900 cases he encountered only one with multiple perforations. Hurst and Stewart¹⁹ observed at postmortem examinations that about 12 per cent of all cases of perforation were multiple. In collective statistics, compiled from large numbers of cases, the incidence of multiple perforations is only about 4 per cent. Multiple perforations usually consist of two such lesions; however, three and even four simultaneous perforations have been reported.

BACTERIOLOGIC EXAMINATION. In about one-half of all cases of perforation the contents escaping from the stomach are sterile; in the remaining half they yield positive cultures. Frequently, they are streptococci (60 per cent), staphylococci (25 per cent), to a lesser extent *Bacillus coli*, diphtheroid, aerogeneous and pneumococci.

SYMPTOMATOLOGY. The symptoms are characteristic depending upon the time which is allowed to elapse between perforation and medical examination and upon the extent and site of perforation, whether the ulcer ruptured into the free peritoneal cavity, some viscus, or an encapsulated space. In about 90 per cent the patients are able to give a history of indigestion and other digestive disturbances ranging from a few days to many months. Most of these patients state that some days

prior to rupture pain and discomfort were aggravated and not relieved by alkalines. In most cases the perforation takes place while the patient is active or after a meal. However, it also occurs frequently in an empty stomach while the patient is asleep.

When the patient is seen immediately after perforation the most striking symptom is pain. It appears suddenly, is very intensive, and spasmodic or constant; it may be generalized or localized. Localized pain is most frequently centered in the epigastrium and then, in order of frequency, in the right lower quadrant, hypogastrium, right upper quadrant, in the back, left upper quadrant and left lower quadrant. In about one-fourth of all cases the patient also complains of referred pain in the shoulders. The order of frequency is ordinarily the right shoulder, both shoulders, or the left shoulder. The pain in the shoulders is due to transmission from the branches of the phrenic nerves in the diaphragm to the main trunk ($C_3 + C_4$) and from there to the clavicular and acromial branches. Since the rupture is most frequently of the prepyloric or duodenal ulcer type, the pain is referred to the right shoulder. Perforation occurs less frequently halfway between the pylorus and the cardia and, therefore, pain in both shoulders is infrequent. Perforation is rarely close to the cardia and hence few cases are observed with pain referred solely to the left shoulder.

Tenderness and rigidity are prominent symptoms. Rigidity is generalized and present in more than 90 per cent of all perforations; it is so pronounced that many clinicians designate it as "boardlike." However, sometimes tenderness and rigidity are localized and occasionally they are absent; in the latter case diagnosis may be extremely difficult.

Nausea and vomiting is present in about 50 per cent of all cases.

The external appearance of the patient is characteristic: pale face, anxious expression, cold perspiration on the face; the nostrils are widened; the skin is cold and clammy; respiration is of the costal type and shallow; the patient being afraid to move lies immobile. The general impression is that of a state of shock as described in many manuals; however, the pulse is only slightly accelerated, is of good quality and the blood pressure is not much lowered; in other words, the patient is rarely in a state of actual shock.

Temperature is normal or subnormal in the first stage; later, when signs of peritonitis appear, the temperature rises. *The pulse* is moderately increased, i.e., about 90. *Respiration* increases to 24 to 30 per minute, is shallow and costal in character. *Liver dullness* is diminished or absent in about 60 per cent. *Free fluid*, is usually, found in cases in which perforation took place at least eight hours prior to examination.

This *primary stage* persists two to four hours and is followed by the *intermediate stage* or *stage of masked peritonitis*. During this stage the symptoms cease to be acute; the pain is lessened, the muscles of the

abdominal wall become softer, the pulse slows down, anxiety is less pronounced and the skin becomes warmer.

If the patient is first seen at this stage by a surgeon with limited experience, the condition may not be recognized until the succeeding stage of *frank peritonitis*. A more experienced surgeon, however, does not overlook the symptoms of masked peritonitis. The abdominal wall is rigid and tender, the patient lies with his legs flexed in the knees and hip joints, and has pain in attempting to change his position. The pelvic peritoneum is *extremely tender*. This may be elicited in a woman by vaginal, in men by rectal examination. The tenderness is due to accumulation of the gastrointestinal contents which escaped into the pelvic cavity and irritation of the pelvic peritoneum. It is noticeable two to three hours after perforation but may never appear if the perforation is small or when it is sealed promptly. Thus tenderness of the pelvic peritoneum is of great diagnostic significance although its absence does not disprove perforation.

Hemorrhage in connection with perforation does not occur frequently. In the past surgeons did not associate these two conditions; in fact, it was a common belief that "bleeding ulcers do not perforate and perforated ulcers do not bleed." However, investigations into a great number of cases have revealed that almost 8 per cent of perforations are accompanied by bleeding, as illustrated by melena or hematemesis, or both. This incidence is observed most frequently in patients in their fifties or sixties. The subject of hemorrhage in perforated peptic ulcer will be discussed later.

LABORATORY FINDINGS. *Blood examination* shows a decided leukocytosis even in the first stage and more so in the second and third stages. The average white cell count reaches 15,000, but cases with a count as high as 40,000 have been reported. The percentage of polymorphonuclear cells averages about 80. Occasionally, leukopenia is found with a white cell count as low as 3,000.

X-ray examination is of great value, especially in doubtful cases. Weinberger,²⁰ in 1908, observed by x-ray accumulation of gas under the diaphragm. Popper,²¹ in 1915, reported a case in which he observed gas under the diaphragm fluoroscopically. He explained the picture as air which escaped from a perforated peptic ulcer. Weiland²² made the same observation in 1915. Lenk,²³ in 1916, called attention to the diagnostic significance of free air in the peritoneal cavity.

Most roentgenologists examine the patient in an upright position. In this position it was found that in 20 to 25 per cent of the cases of rupture there was no air in the abdomen. Vaughan and Brams,²⁴ in 1925, reported fifteen cases of anatomically proved perforations of gastric or duodenal ulcer, in thirteen of which air was shown roentgenologically. Finsterbusch and Gross,²⁵ in 1932, stated that they gave preference to

the left lateral decubitus position because, in this way, even a very small amount of gas can be observed in contrast to the dense shadow of the liver, the diaphragm and lateral wall of the abdomen. Vaughan and Singer,²⁶ in 1933, studied ninety-seven cases of perforated gastric and duodenal ulcer, and came to the conclusion that the most favorable position in which to detect air is that of the patient lying on his left side. Williams and Hartzell,²⁷ in 1940, reported a series of sixty-eight patients with perforated gastroduodenal ulcer who were fluoroscoped in the left lateral decubitus position; failure of detection was reduced from 25 per cent in upright examination to 11 per cent in the left lateral decubitus position.

DIAGNOSIS. A history of digestive disturbances prior to an acute attack, the preceding aggravation of the symptoms which were not relieved by dietary measures, severe abdominal pain, board like rigidity of the abdominal muscles, the characteristic expression of the face, and detection of air under the diaphragm, all these factors render a clear diagnosis possible in 90 per cent of all cases. However, a proper diagnosis becomes difficult in atypical cases, e.g., when the ulcer ruptures into a hollow viscus, or when the contents escape into the cecal region along the lateral border of the ascending colon, or the original opening closes spontaneously by means of an omental plug. Some of the erroneous diagnoses are, in the order of their frequency, acute appendicitis, acute cholecystitis, intestinal obstruction and acute pancreatitis, also acute mesenteric thrombosis and ectopic pregnancy. Nevertheless, such errors in diagnosis are not the cause for fatal consequences as these conditions require surgical intervention. However, there are some non surgical conditions which may be mistakenly diagnosed as acute perforated ulcer and a laparotomy would harm the patient seriously. Such conditions are: pneumonia, coronary thrombosis, renal or biliary colic, gastric crises of tabes and acute alcoholism.

Differential Diagnosis. In differentiating perforated ulcer from any other condition, the task is facilitated if any extraperitoneal process is primarily excluded. In an intra-abdominal affliction pain and tenderness are associated, the latter being intense at one particular point of the abdomen; the pain is aggravated by a change of position. Bearing this in mind it is comparatively easy to differentiate intra-abdominal from extra-abdominal diseases, such as central pneumonia or coronary thrombosis.

In **coronary thrombosis** the pain does not appear in a flash; there is no abdominal muscular rigidity and no point of maximum tenderness. Pain and tenderness are not necessarily associated and pain is not aggravated by movement. Fluoroscopy of the patient, in a left lateral position, reveals air in 95 per cent of cases of perforated ulcer.

In **right side pneumonia** there may be great epigastric pain and

rigidity of the abdominal muscles; the patient may lie prostrate. There is fever, a high pulse rate, rapid respiration; digital examination of rectum or vagina does not reveal tenderness of the pelvic peritoneum; there is no liver dullness.

Differentiation from biliary colic. Severe pain, moderately fast pulse, and the expression of the face may be identical in biliary colic and peptic perforation. However, the preceding history is different. In biliary colic pain and jaundice are reported by the patient, the pain radiating to the subscapular region. The abdominal wall is not or only moderately rigid; the patient is restless and tries to find a more comfortable position. Liver dullness is not diminished and the pelvic peritoneum is not tender. Roentgenoscopy reveals free air in about 90 per cent in cases of perforated ulcer; there is none shown in cases of biliary colic.

Differentiation from renal colic. The history may reveal renal attacks or hematuria, or passing of small stones (gravel). The patient may complain of pain radiating to the testicle. The abdomen is not rigid nor is there any fear of moving; but decided restlessness and efforts to find a more comfortable position exist.

Gastric crises of tabes dorsalis may give a picture of excruciating pain and collapse. However, there may be a history of similar attacks in the past. Physical examination may show soft abdominal muscles, absence of patellar reflexes and the Argyll-Robertson sign. There is no tenderness of the peritoneum upon rectal examination and liver dullness is not diminished.

Acute pancreatitis may be readily mistaken for perforated gastric or duodenal ulcer, although the pain is more intense, rigidity more confined to the epigastric area and not as generalized as in perforation of gastric or duodenal ulcer. Glycosuria, a positive Loewi test and an increase in the diastase contents in the urine point to pancreatitis, in addition to bilateral lumbar pain and slight jaundice. There is no free air observed in the peritoneum.

Intestinal obstruction may resemble perforation in cases of strangulation of a bowel. In both conditions there is extreme pain, vomiting, collapse, but in obstruction the abdominal wall is soft, at least in the early stage. The x-ray picture is so characteristic of each of these conditions that this alone prevents diagnostic errors.

Ruptured ectopic pregnancy may be accompanied by severe abdominal pain, vomiting and shock. The history reveals irregular menstruation; the patient is extremely anemic which can be especially observed in pale lips, nails and conjunctiva. There is only mild tenderness and slight rigidity in the lower part of the abdomen. It has been pointed out previously that free air is found in about 90 per cent of all cases of ruptured peptic ulcer and, therefore, its presence at once rules out a diagnosis of ectopic pregnancy.

PROGNOSIS. Statistics have revealed that the rate of immediate mortality in cases of perforated gastric or duodenal ulcer is high, ranging from 10 to 40 per cent, 25 per cent being a conservative estimate. The fatal outcome is determined by several factors, such as the time elapsed between perforation and operation, sex, age, site and extent of perforation, whether it ruptured into the free peritoneal cavity or viscus, preoperative management, type of anesthesia employed, type of operation performed, and the general constitution of the patient. However, there has been a marked tendency toward lowered mortality. The time elapsed between perforation and operation is probably the most decisive single factor. Studies of a large number of cases have shown that the mortality is doubled when operation is performed six to twelve hours after perforation, trebled after twelve to twenty-four hours; it is estimated to be almost five times as great if the operation is done twenty-four hours after perforation as compared with operations performed within six hours.

Sex. The mortality among women is relatively higher than among men. Considering the fact that in peritonitis the mortality in women is lower than in men, it is surprising that women present a higher mortality in perforated ulcer than men. No satisfactory explanation for this apparent peculiarity is available.

Age plays a great prognostic rôle. The rate of mortality is the lowest under thirty, is almost doubled between the ages of thirty and fifty, and increases fourfold between fifty and seventy.

Site of Perforation. It has been found that mortality is higher in perforated gastric than duodenal ulcer. This may be explained by several factors. In gastric perforation the opening is usually larger and, therefore, spillage is more abundant; the contents are more acid and, thus, more irritating and the shock more increased; it occurs more frequently in older patients who are in the group with a higher rate of mortality.

ANESTHESIA. There is no unanimous opinion as to what constitutes the most effective anesthesia. If the blood pressure is above 130, spinal analgesia should be employed because the bowels are almost motionless and do not crowd the operative field. However, if the blood pressure is low, spinal analgesia is not advisable because it may reduce the blood pressure to the shock level. In cases of low blood pressure general or local anesthesia should be administered, using ether, ethylene or cyclopropane in general anesthesia. The disadvantages of general anesthesia is that strong inspiratory movements during the induction period compress the diaphragm and, by pumping the stomach to some extent, expel some of the gastric or duodenal contents into the free peritoneal cavity.

Local anesthesia may be employed in cases in which the perforation is immediately located as, otherwise, the manipulations involved in its search are very painful and exhausting to the patient. Until the surgeon

opens the abdomen he is in no position to determine whether or not he will have difficulties in locating the ulcer; therefore, local anesthesia is not advisable unless the patient is in too bad a condition to risk any other type of anesthesia. This is why statistical reviews record the highest rate of mortality in the application of local analgesia. Consequently, the erroneous deduction is frequently made that local anesthesia involves the greatest risk, thereby not allowing for the fact that it is usually carried out on patients in a very bad general condition.

TYPE OF OPERATION. All being equal, the simpler the operation the less is the immediate operative mortality. Nevertheless, many other factors have to be taken into consideration when selecting the type of operation. This will be discussed later in the "Evaluation of Different Operative Procedures."

TREATMENT. The therapy of perforated ulcer is surgical. In very rare instances patients have recovered without an operation but these were cases in which an omental plug spontaneously sealed the opening immediately after perforation, or when the ulcer perforated into a hollow viscus, such as the gallbladder or jejunum. Such cases are exceptional and it should be borne in mind that the mortality of non-operative cases is almost 100 per cent.

Keeping in mind that the shorter the period between perforation and operation the lower the mortality rate, the least time should be spent on preoperative treatment. It should be confined to injecting morphine hypodermically, administering coramine or another heart stimulant, and introducing normal saline and glucose, intravenously or subcutaneously. The patient should be taken to the operating room without delay, regardless of the amount of saline solution that has been administered. If not much of it was given, it may be continued in the operating room while proceeding with the operation.

Evaluation of Different Surgical Methods

Several procedures are employed, such as simple closure with or without supplementary gastroenterostomy, excision of the ulcer and pyloroplasty, and partial gastrectomy. In the United States and England simple closure is most frequently employed whereas most surgeons on the continent of Europe prefer partial gastrectomy.

SIMPLE CLOSURE is in itself a simple procedure. It was first performed by Mikulicz in 1880 in the treatment of a perforated peptic ulcer; since then it has become the most widely used method. It is simple in its technic and requires very little time; it does not necessitate intricate instruments nor special training on the part of the assistant. Even a surgeon with limited experience may employ it and the method is, therefore, very useful in emergency surgery.

There are several minor variations in its technic: (1) closure by

through-and-through interrupted sutures, two or three in number, penetrating through the entire thickness of the gastric wall and followed by a second row of sero-serosa sutures (Lembert) (Fig. 44, A). This method is employed in cases of extensive perforation and when the ulcer has indurated edges. (2) Seromuscular purse-string suture followed by a few Lembert sutures (Fig. 44, B). This method is used in small perforations and when the ulcer is not indurated. (3) Omental graft over the opening

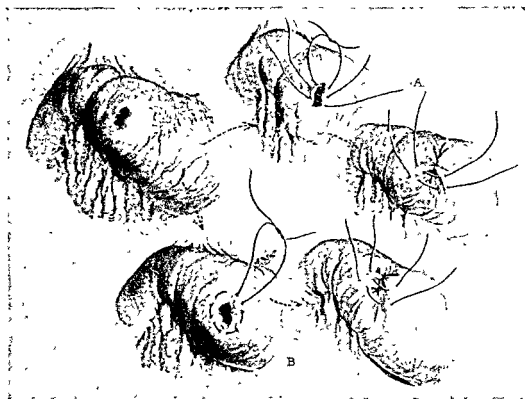


FIG. 44. Perforated gastric ulcer. Simple closure.
(For details, see the text.)

after closure as described in (1) and (2) (Fig. 45, A). (4) Use of an omental plug to close the perforation. This method is employed in cases with an extensive indurated area in which closure by mere suturing would not be secure (Fig. 45, B). We do not consider this method advisable for the reason that the surgeon should resort to excision with pyloroplasty in cases in which the area is so extensively indurated that simple sutures would not ensure safe closure. (5) Cellan-Jones method. This method consists in plugging the opening with omentum. Its technic in detail is described later.

The advocates of simple closure prefer this method to any other for the following reasons: (a) Acute perforated ulcer is an emergency. The patient may be in a state of shock or greatly weakened; the operative

area is contaminated. Under these conditions the surgeon's sole responsibility toward the patient is to close the opening to prevent almost certain death due to peritonitis. Any other treatment for the patient's cure has to be postponed until the patient recovers to a point at which he is enabled to withstand a more extensive operation. This is particularly emphasized by the growing tendency to lower the rate in operative mortality by performing a series of fractional operations. For instance, in thyrotoxicosis, when the patient is considered a poor operative risk for

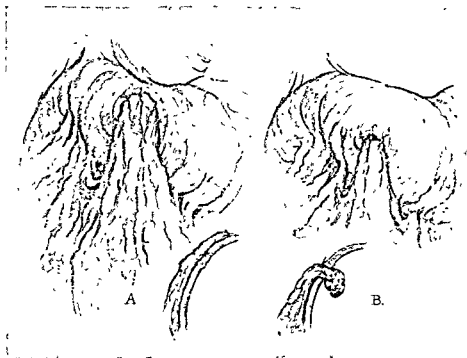


FIG. 45. Perforated gastric ulcer. Closure with omentum. A. Omentum over the opening. B. Omental plug.

a bilateral thyroidectomy, the surgeon confines himself to the removal of one lobe and operates upon the other when the patient's condition permits it. When it appears likely that even the removal of one lobe may endanger the patient, the surgeon resorts to ligation of the vessels of the upper and lower poles on the same side or of the upper pole alone. Experience has shown this to be advisable because the rate of mortality has been thus lowered. The same applies to other organs: when a patient seems unsuited for a gastrectomy in one stage, it is carried out in two stages by performing gastroenterostomy in the first stage and removing the affected part of the stomach, with closure of the duodenal and gastric stumps in the second stage.

In about 60 per cent of all cases the simple closure not only takes care of the immediate emergency but, actually, has a curative action;

the patient remains free from symptoms. In the remaining 40 per cent of patients only one-third require re-operation at a later time, the others faring well under medical care. In other words, only about 15 per cent of all patients with perforated gastroduodenal ulcer in whom perforation is closed by simple suture, need to be re-operated upon. If this is done at the optimum time, it is to the greater advantage of the patient than during an acute emergency.

SIMPLE CLOSURE WITH GASTROENTEROSTOMY. This method was first employed by Braun⁸ in 1897. Later, it was advocated by Hartman, Moynihan and others, for the following reasons: (1) A perforated ulcer, in the majority of cases, lies close to the pyloroduodenal junction, and closure of the opening may constrict the pyloric outlet. (2) Even when the closure of the perforation does not appreciably constrict the pyloric canal the mere passage of food along the area where the perforation was closed will keep the suture line under tension and, thus, retard healing by peristaltic contraction of this part. By doing gastrojejunostomy, with the opening proximal to the perforated point, the tension on the closed area is diminished, thus enhancing the healing process.

The opponents of this method raise the following objections: The method considerably prolongs the operation and, thereby, increases the immediate mortality rate. Patients with perforated peptic ulcer have an increased acidity. Gastroenterostomy involves the risk of a peptic ulcer being produced at the site of the stoma. Ulcers at this locality are very troublesome.

EXCISION OF ULCER AND PYLOROPLASTY. This method of treating perforated peptic ulcer was introduced by Dowden¹² in 1909; however, it never gained popularity. The following reasons were given for its employment: (1) an indurated infectious lesion is removed; (2) normal tissues heal more rapidly than indurated structures; (3) the "unphysiologic" gastroenterostomy is replaced by a physiologic procedure, thus eliminating gastroenterostomy and the possibility of formation of peptic gastrojejunal ulcer; (4) the period of operation is not appreciably increased. Nevertheless, this method has not been widely accepted but we believe it to be a rational procedure because it does not require much time, obviates the necessity of any other supplementary procedure in cases in which the mere closure of perforation would constrict the pyloric outlet; it also removes indurated infectious tissues. However, if the induration is so extensive that excision of a large amount of tissue becomes necessary, the operation is mechanically not applicable.

PARTIAL GASTRECTOMY is advocated by Continental European surgeons. Haberer,¹¹ in 1919, recommended it as an operation of choice for perforated gastric or duodenal ulcer. Judine,¹⁶ in 1939, reported a personal series of 937 cases with the unusually low rate of mortality of 8.9 per cent.

The followers of gastrectomy advocate its use for the following reasons: It removes not only the pathologic area but also the entire ulcer-bearing and acid-producing region. Thus, it cures not only palliatively but radically 80 per cent of all cases, whereas, by mere closure of the perforation, only 60 per cent are cured. Of the latter group about 25 per cent require medical attention at a later date and 15 per cent have to be re-operated upon.

On the other hand, the opponents of gastrectomy point out that the rate of immediate mortality after gastrectomy is considerably higher than after simple closure. The fact that some surgeons who perform this operation for perforated ulcer are able to show a low rate of mortality does not disprove the claim of the opponents that mortality after gastrectomy is always higher than after simple closure. The reason why some skilled surgeons have been able to show a low mortality rate is that they have been working in superior university clinics of large cities. If this method were employed in small community hospitals where frequently only a casual operator is available, gastrectomy would have disastrous results. As in many cases of perforation streptococci are present, it is advisable to use sulpha derivatives in the abdominal cavity and abdominal wall. Five to 10 gm. of sulfanilamide or sulfathiazole powder is placed in the peritoneal cavity around the lesion, and about 3 to 5 gm. in the abdominal wall.

Many reports show that the local application of drugs of the sulfa group substantially reduces the mortality rate. Although the drugs are applied locally, its action is systemic since determination of sulfa drugs in the blood shows that, after placing 10 gm. of the powder in the peritoneal cavity and 5 gm. in the abdominal wall, its maximum concentration in the blood is reached within four hours and amounts to about 6 mg. per 100 cc., of blood. After four hours it begins to diminish and disappears within twenty-four to forty-eight hours. It is also advisable to administer this drug after operation intravenously until the temperature drops to normal.

DRAINAGE. We do not drain the peritoneal cavity after operations on the stomach. Prior to introduction of the sulfa drugs we used to place a Penrose drain into the abdominal wall down to the sutured peritoneum with the loose end left outside between two interrupted skin sutures in those cases in which we were suspicious of contamination of the abdominal wall. Since the sulfa drugs became available, we introduce 5 gm. of powdered sulfathiazole into the abdominal wall and close it without drainage.

POSTOPERATIVE TREATMENT. When the operation is completed the patient is given 2,000 to 3,000 cc. of normal salt solution, or 10 per cent glucose by venoclysis, or 5 per cent hypodermically every twenty-four hours. Undoubtedly, venoclysis is more convenient to the patient than

hypodermoclysis and it should, therefore, be employed wherever the patient can be under constant close supervision for the possibility of a fatal overloading of the heart due to the intravenous administration of fluids. With hypodermoclysis an overloading of the heart, by rapid absorption, does not occur and, therefore, its use is advisable where adequate facilities are not available. The amount of fluid given each time is 1,000 cc.

Furthermore, a Levine tube is inserted into the stomach and kept there for twenty-four to forty-eight hours. If symptoms of peritonitis appear, the tube is retained for a longer period. The patient is given morphine, gr. $\frac{1}{2}$, two to three times daily. When there are no complications, the Levine tube is removed after forty-eight hours and the patient takes liquid nourishment, e.g., milk, cream, and later is placed on an ulcer diet. Complications which may arise are treated accordingly.

COMPLICATIONS arise in a large number of cases of perforations; according to some statistics as much as in 50 per cent. In the order of frequency they are: pulmonary complications, peritonitis, wound infection, subphrenic abscess; these compose about 90 per cent of all complications. Furthermore, there is gastric hemorrhage, pelvic abscess, intestinal obstruction, duodenal fistula, gastric fistula, thrombophlebitis and otitis media.

CAUSES OF DEATH. The causes of death in the order of frequency are bronchopneumonia, lobar pneumonia, peritonitis and, occasionally, embolism and gastric hemorrhage.

Operative Surgical Technic

SIMPLE CLOSURE. In cases of small perforation and when the ulcer is not indurated the following technic may be applied:

First step: The abdomen is opened by a right paramedian incision. The abdominal cavity is cleansed of gastric contents.

Second step: Two or three interrupted sutures of chromic catgut No. 1 are inserted through the edge of each lip so as to penetrate the entire thickness of the gastric wall (Fig. 44, A).

Third step: The first row of sutures is reinforced by a second layer of interrupted sutures of silk or linen which penetrate only the seromuscular layer (Fig 44, A).

Fourth step: The abdomen is closed without drainage.

Another method of treatment of small perforated and not indurated ulcers is as follows:

First step: As in the previously described method.

Second step: A purse-string seromuscular suture of silk or linen is made with the ends tied together; this is followed by a second layer of two or three interrupted seromuscular sutures of silk or linen (Fig. 44, B).

Third step: Same as fourth step in previously described operation.

In cases of large perforation with the gastric wall in the vicinity of the perforation being indurated, the following method may be employed:

First step: The abdomen is opened as in the preceding methods.

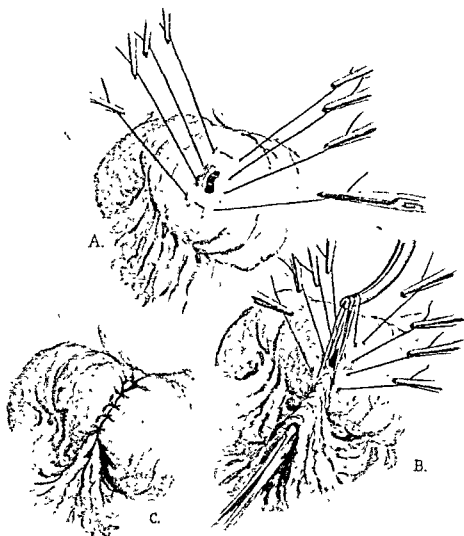


FIG. 46. Closure of perforated gastric ulcer by Cellan-Jones method.

Second step: Two or three chromic catgut No. 1 interrupted sutures are made through the entire thickness of the gastric wall, and the ends are tied with moderate force to prevent the threads from cutting through the gastric wall.

Third step: A small piece of omentum, *not detached* from its main bulk is placed over the perforated area and fixed to the anterior gastric wall

by several interrupted sutures of plain catgut No. 1 (Fig. 45, A).

Fourth step: The abdomen is closed without drainage.

If, however, the ulcer in the vicinity of the perforation is indurated to such an extent that the edges cannot be even loosely approximated, the following technic may be employed:

First step: As in the preceding operations.

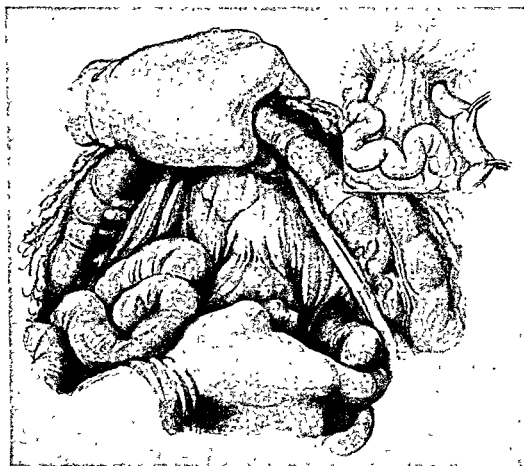


FIG. 47. Posterior gastroenterostomy. Location of the first jejunal loop.

Second step: A piece of the omentum, not detached from the mass, is plugged tightly into the perforation and fixed to the adjoining margin of the ulcer by means of a fine Pagenstecher linen thread (Fig. 45, B). This is repeated in several places.

Third step: The abdomen is closed without drainage.

CELLAN-JONES METHOD. First step: The abdomen is opened as in the preceding operations.

Second step: Two through-and-through sutures are inserted in such a way that each thread enters into the lumen from without, at a distance of one-quarter inch from the margin of the perforation, leaving the lumen from within at a distance of one-quarter inch from the other lip of per-

foration. Each thread is clamped by an artery forceps. A seromuscular suture is placed above the perforation and one below it. The ends of the threads are clamped by means of an artery forceps (Fig. 46, A).

Third step: The central part of each thread is raised into an arch, and a piece of omentum, held by a forceps, is placed over the perforation; the ends of the threads are tied together (Fig. 46 B and C).

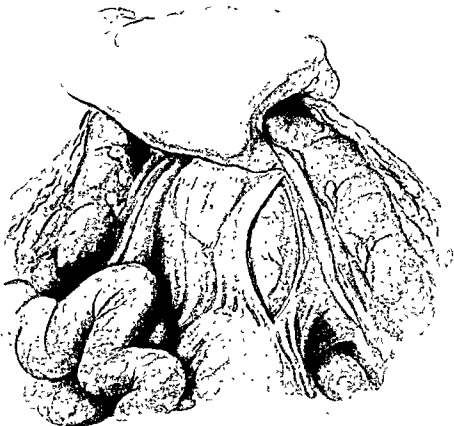


FIG. 48. Posterior gastroenterostomy. A rent is made in transverse mesocolon

Fourth step: The abdomen is closed without drainage.

SIMPLE CLOSURE WITH GASTROENTEROSTOMY. The merits and disadvantages of this method were discussed earlier. Its technic is the following:

First step: The abdomen is opened; the operative field examined, and the gastric contents are sponged from the peritoneal cavity.

Second step: The opening in the stomach is closed by the means described in the other methods.

Third step: Gastroenterostomy is performed as posterior or anterior procedure with enteroanastomosis.

Posterior gastroenterostomy involves the following steps: (1) The colon is lifted with the left hand and the transverse mesocolon is put on a moderate stretch (2) The first jejunal loop is located by means of the Woelfler-Socin method: The right hand is placed on the vertebral column at the base of the transverse mesocolon so that it rests on the body of the second lumbar vertebra with its index finger sliding to the left of



FIG. 49. Posterior gastroenterostomy. A portion of posterior gastric wall is pushed downward through the opening in the transverse mesocolon.

this vertebra. The loop which lies between the index finger and the left side of the second lumbar vertebra is the first loop of the jejunum. A segment of the first jejunal loop, starting two inches from the duodeno-jejunal junction and extending four inches, is grasped (Fig. 47), the contents are squeezed from it and the loop is compressed axially by an intestinal clamp which is tight enough to prevent an escape of intestinal contents but not so tight as to shut off circulation. (3) An opening is made in the transverse mesocolon, perpendicularly to the long axis of the colon (Fig. 48). This opening is at the left of the main stem of the

middle colic artery, an area with comparatively few blood vessels ("avascular area of Riolan"). (4) A part of the posterior wall of the stomach is pushed downward through the opening in the transverse mesocolon (Fig. 49) in the following manner:

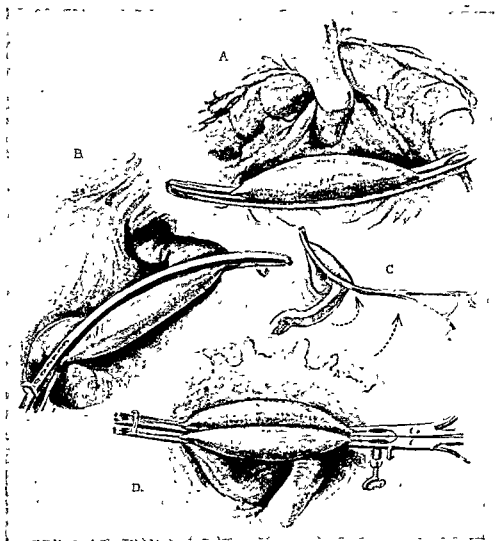


FIG. 50 Posterior gastroenterostomy. A. Clamping a gastric fold. B. Clamping a jejunal fold. C. Rotation of the jejunal fold. D. Gastric and jejunal folds are placed side by side.

The surgeon places the tip of his little finger of the left hand on the anterior wall of the stomach close to the angular notch. The index finger is placed on a vertical line running from left end of the cardia to the greater curvature, at a distance of one inch from the greater curvature. The middle and fourth fingers are placed between the little and index fingers. Then the four fingers push the stomach through the rent in the

mesocolon and, thereby, the posterior gastric wall appears below the transverse mesocolon (Fig. 49). The ends of the protruding part of the stomach are held at each end with Allis forceps. The posterior half of the

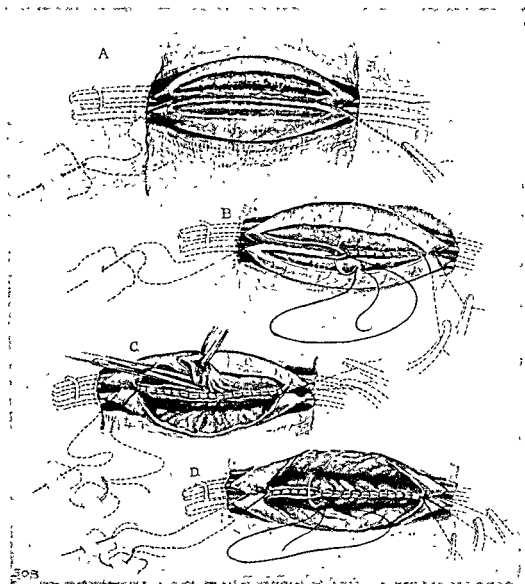


FIG. 51. Posterior gastroenterostomy. Technic of anastomosis A. Sero-serosa suture is inserted. Ligation of small blood vessels traversing across the mucosa. Mucosa is not opened yet. B. Placing seromuscular-seromuscular sutures. The mucosa is not opened yet. C. Incision of the mucosa. D. Suturing the mucosa of the stomach to that of the jejunum.

circumference of the rent in the transverse mesocolon is sutured to the gastric wall (Fig. 49). The protruded part of the stomach is then grasped by an intestinal clamp in such a manner that its handle is directed toward the left shoulder and its tip toward the right hip joint (Fig. 50, A). The jejunal fold is held in such a way that the handle of the clamp is directed toward the right hip joint and its tip toward the left shoulder

(Fig. 50, B). The clamp holding the jejunum is then turned 180 degrees so that the handles of both clamps lie side by side (Fig. 50, C and D). The proximal part of the jejunum lies, therefore, adjacent to the part of the gastric fold close to the lesser curvature, and the distal jejunal end

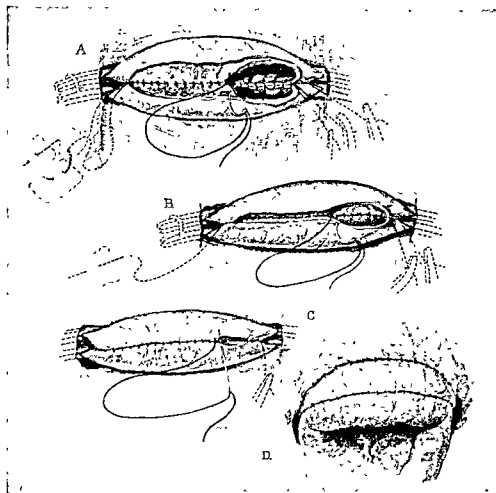


FIG. 52. Posterior gastroenterostomy. A. Suturing the gastric mucosa to that of the jejunum anteriorly. B. Suturing the muscular layer of the stomach to that of the jejunum. C. Suturing the serosa of the gastric wall to that of the jejunum. D. The anterior lip of the transverse mesocolon is sutured to the anterior gastric wall.

lies at the side of the gastric portion closer to the greater curvature. (5) Anastomosis is carried out between the gastric and jejunal folds in the same manner as a lateral intestinal anastomosis between two segments of a bowel (Figs. 51 and 52). (6) When the anastomosis is completed the anterior lip of the opening in the transverse mesocolon is sutured to the stomach close to the suture line of the anastomosis, thus preventing a loop of small bowel from slipping into the ring and being strangulated (Fig. 52, D).

The technic of anterior gastroenterostomy is as follows: (1) After the abdomen is opened the transverse colon is lifted as described in previous operation. (2) The location of the first jejunal loop is done exactly in the same way as described above; however, instead of utilizing a segment of



FIG. 53. Anterior gastroenterostomy with enteroanastomosis.

bowel two to three inches away from the duodenojejunal junction it is utilized a loop situated at a distance of twelve to sixteen inches away from the same junction. The length of the segment is four inches—the same as in case of posterior gastrojejunostomy. (3) A portion of the anterior wall of the body of the stomach, close to the greater curvature and parallel to it, is grasped axially for a length of four inches in an intestinal

clamp. (4) The gastric and intestinal segments are laid side by side isoperistaltically. (5) A lateral anastomosis is carried out in the same manner as in two loops of bowel. (6) A lateral intestinal anastomosis is added between the afferent and efferent loops of the bowel (Fig. 53). (7) The abdomen is closed without drainage.

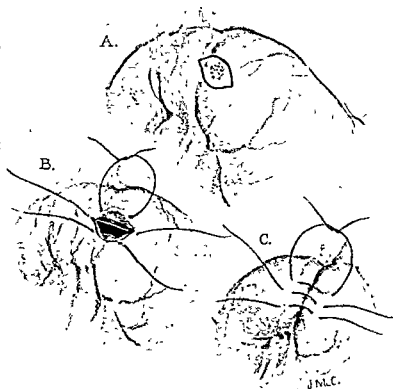


FIG. 54. Excision of a perforated ulcer with a pyloroplasty.
 A. Excision of the ulcer.
 B. Placing the first layer of through-and-through sutures.
 C. Placing the second layer of sero-serosa sutures.

EXCISION OF PERFORATED ULCER WITH PYLOROPLASTY. The claims advanced in connection with this method were discussed earlier; its technic is the following:

First step: The abdomen is opened as in the preceding methods; the abdominal cavity is cleansed from the escaped gastric contents.

Second step: A rhomboid or elliptical incision is made around the perforated area (Fig. 54, A). The excised part should include all indurated edges.

Third step: The opening is closed in a direction perpendicular to the axis of the pyloric canal. The first layer penetrates the entire thickness of the gastric and duodenal walls (Fig. 54, B), and the second layer is sero-

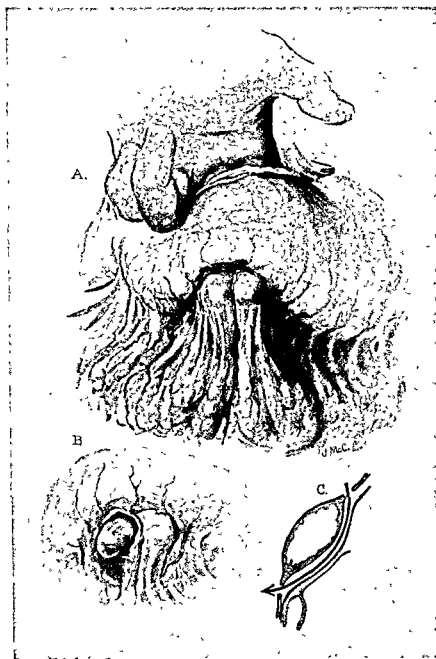


FIG. 55. Partial gastrectomy. First step in the division of the gastrocolic ligament.

muscular (Fig. 54, C). As previously pointed out, the operation is feasible provided the perforation is close to the pyloroduodenal junction and the indurated area is small; otherwise, it is not practicable from a

mechanical point of view. In perforations in which the ruptured gastric ulcer is at some distance from the pyloroduodenal junction, excision of

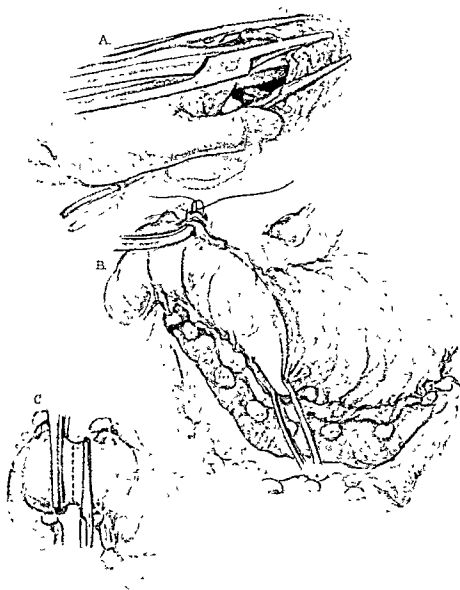


FIG. 56 Partial gastrectomy. A. Division of the gastrocolic ligament between clamps. B. The right and the left gastroepiploic arteries are divided and the divided ends tied already. A clamp is seen beneath the right gastric artery preliminary to its ligation. C. Division of the first portion of the duodenum between two clamps.

the ulcer and suturing of the stomach in two layers is relatively simple procedure.

PARTIAL GASTRECTOMY is performed with the Billroth I or Billroth II method. Whereas, in a gastric perforation both Billroth methods may

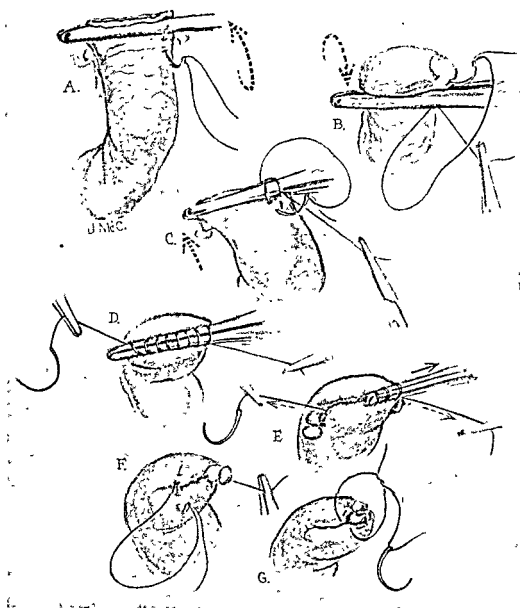


FIG. 57. Partial gastrectomy. Closure of the duodenal end by Parker-Kerr method. (For detailed description, see the text).

be employed, the Billroth I may not always be possible in duodenal perforation. The following is a description of the Billroth II operation and some of its modifications:

BILLROTH II AND ITS MODIFICATIONS.*

a. Polya method (partial gastrectomy plus closure of the duodenal end plus end-to-side implantation of the remaining portion of the stomach into the first jejunal loop retrocolically).

First step: The abdomen is opened by a left paramedian incision.

Second step: Division of the gastrocolic ligament for the required length between clamps (Figs. 55, 56A).



FIG. 58. Partial gastrectomy. Ligation of the left gastric artery.

Third step: Severing of the right gastroepiploic artery between two ligatures (Fig. 56, B).

Fourth step: Severing of the left gastroepiploic artery between two ligatures (Fig. 56, B).

Fifth step: Division of the right gastric artery between two ligatures (Fig. 56, B).

Sixth step: Division of the gastrohepatic ligament between clamps.

Seventh step: Division of the first portion of the duodenum between two crushing clamps (Fig. 56, B).

* For evaluation of different methods, the indications for their employment and details of technic, the reader is referred to Spivack's "The Surgical Technic of Abdominal Operations" Charles C Thomas, Publisher, Springfield, Illinois, 1946. This presentation is confined to only a few methods.

Eighth step: Closure of the duodenum over the lower clamp by the Parker-Kerr method (Fig. 57).

Ninth step: Double ligation of the left gastric artery (Fig. 58).

Tenth step: An opening is made in the transverse mesocolon, to the left of the middle colic artery; the first jejunal loop is located by the

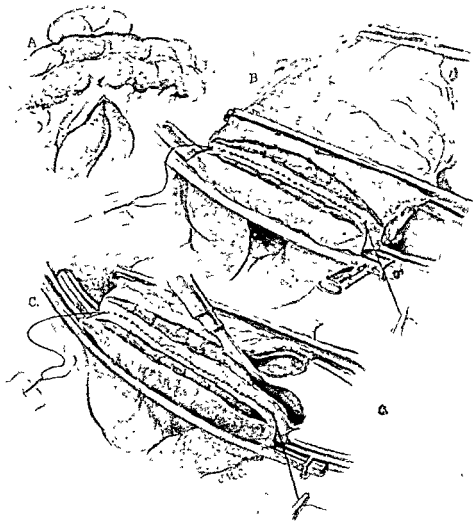


FIG. 59. Partial gastrectomy. A. The first jejunal loop is carried through a rent in transverse mesocolon and placed along the posterior gastric wall. B. Seromuscular-seromuscular suture connecting the jejunum and the posterior gastric wall. C. The stomach is being cut away between the Payr clamp and the seromuscular suture line.

Woelfler-Socin method, and carried through this opening and placed along the posterior wall of the stomach so that the proximal end of the jejunal loop lies at the lesser curvature and the distal at the greater curvature (Fig. 59).

Eleventh step: A seromuscular suture connects the jejunal loop with the posterior wall of the stomach, along a line running one-quarter inch

proximal to the line connecting the ligated left gastric with the left gastroepiploic arteries. The stomach and jejunum are clamped behind the suture line by means of intestinal clamps (Fig. 59, B).

Twelfth step: A Payr or other crushing clamp is placed upon the

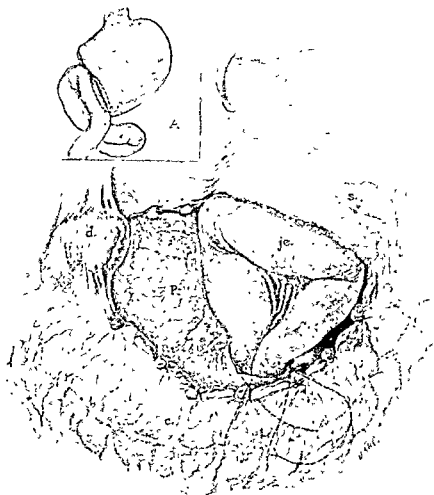


FIG. 60 Polya method (final view). Insert A. Hoffmeister-Finsterer's modification.
s. Stomach. je. Jejunum. c. Colon P. Pancreas. d. Duodenum

stomach about one inch distal to the seromuscular suture line. A linear incision is made through the jejunal wall, one-sixth inch from the seromuscular suture line. The stomach is cut away by a linear incision between the Payr clamp and the seromuscular suture line, at a distance of one-third inch from this line (Fig. 59, C).

Thirteenth step: A lateral anastomosis is completed between the

jejunum and the remaining portion of the stomach (Fig. 60).

Fourteenth step: The stomach is sutured to the circumference of the transverse mesocolon to prevent a loop of bowel from slipping into the ring of the transverse mesocolon.

Fifteenth step: The abdomen is closed without drainage.

b. **HOFFMEISTER-FINSTERER MODIFICATION OF THE POLYA OPERATION:** First to tenth steps same as in the Polya operation:

Eleventh step: A seromuscular suture connects the first jejunal loop with the posterior wall of the stomach along a line running one-quarter inch proximal to the line connecting the ligated left gastric with the left gastroepiploic arteries. The length of this suture-line is four inches; it starts at a point four inches from the greater curvature. In other words, only the lower four inches of the line connecting the ligated points of the left gastric and left gastroepiploic arteries are utilized for the anastomosis (Fig. 60).

Twelfth step: An intestinal clamp grasps axially the loop of the jejunum behind the seromuscular suture line; another intestinal clamp grasps the stomach behind the seromuscular suture line, while care is taken that the entire length of the line extending from the lesser to the greater curvature is within the grasp of the forceps which should be placed one inch proximal to the seromuscular suture line.

Thirteenth step: A crushing clamp is placed across the stomach in front of the seromuscular suture line, at a distance of one-third inch from it and extending from the greater to the lesser curvature. The stomach is cut off between the seromuscular suture line and the clamp close to it.

Fourteenth step: The portion of the stomach between the point of the lesser curvature and the upper point of the seromuscular suture is closed by a continuous, through-and-through suture of chromic catgut No. 2.

Fifteenth step: An incision is made through the wall of the jejunum at a distance of one-sixth inch from the seromuscular suture, and a lateral anastomosis is performed between the jejunum and the lower four inches of the stomach which has not been closed in step 14. The thread used for the seromuscular suture in step 11 is now running upward and, after reaching the upper end of the anastomotic line, continues upward on the part of the stomach above the anastomosis, thus peritonizing it.

Sixteenth step: The edges of the transverse mesocolon are sutured all around the stomach.

Seventeenth step: The abdomen is closed without drainage.

c. **BILLROTH II METHOD** (Partial gastrectomy plus closure of the duodenal end plus closure of the stomach end plus posterior gastroenterostomy) (Fig. 61). This is the earliest type of gastrectomy. Though it is

not used as often as Polya method, it still is used from time to time. Its technic is as follows:

Steps 1-9. The same as in Polya method.

Tenth step: Placing of two intestinal clamps across the stomach: one distal to the ligatures on the left gastric and left gastroepiploic arteries, and the other proximal to the same arteries. Cutting of the stomach between the two clamps.

Eleventh step: Closure of the severed end of the stomach, which is occluded by the proximal intestinal clamp; closure is made in two layers:

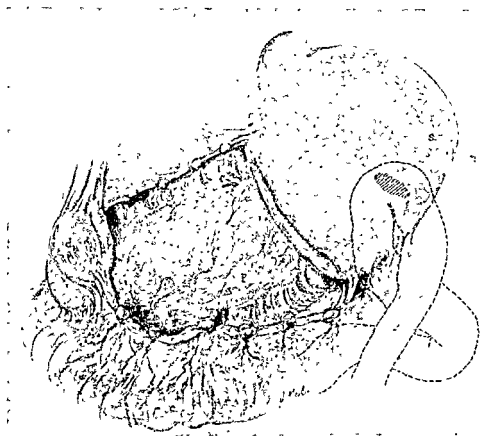


FIG. 61. Billroth II method (final view)

the first by through-and-through sutures and the second in a sero-muscular row.

Twelfth step: Making a rent in the avascular area of transverse mesocolon in the same location and manner as described for posterior gastroenterostomy; location of the first jejunal loop by the Woelfler-Socin method and grasping it axially by means of an intestinal clamp.

Thirteenth step: Protrusion of the posterior wall of the stomach through the opening in the transverse mesocolon and performance of the lateral anastomosis between stomach and bowel are carried out in the same manner as described in the technic of posterior gastroenterostomy.

BLEEDING PEPTIC ULCER

General Remarks

Bleeding occurs in 20 to 30 per cent of all peptic ulcers. It varies in intensity from mere oozing, which can be detected only by a chemical examination for occult blood, to a profuse hemorrhage which may be fatal within a short time. Severe hemorrhage may lead to hematemesis, melena or both; it may show other symptoms of excessive loss of blood, such as rapid and small pulse, pallor of skin and mucous membranes, and the like. The incidence of bleeding peptic ulcers, its clinical manifestations and course vary considerably with different surgeons and clinics. This may be explained by the fact that some clinics place a patient with the same degree of bleeding into different groups. Moreover, the different types of surgical treatment of such patients adds to the difference in the mortality rate.

Those surgeons who consider mere oozing as "bleeding" report a high percentage of bleeding ulcers and a lower mortality rate than those who classify bleeding only when it is manifested by hematemesis or melena. There is a widespread impression, not only among the laity but also the profession, that patients seldom die from a hemorrhage in peptic ulcer. Statistics do not confirm this contention but show that 5 per cent of individuals treated for peptic ulcer die from hemorrhage. There is also a widespread belief that bleeding ulcers do not perforate and perforated ulcers do not bleed; however, this claim is also erroneous. The literature reveals that in a great number of cases almost 8 per cent of all perforations are accompanied by bleeding ulcer prior to the accident or following it. However, it should be borne in mind that bleeding is more frequent in older than in younger patients with peptic ulcer perforation. Statistics show that, while perforation of peptic ulcer occurs most frequently in the age group of thirty to forty, the accompanying bleeding is most frequent in the age group between fifty and sixty years. This increase with advancing age is due to the more frequent presence of arteriosclerosis and hypertension in the older group. The incidence of hemorrhage is by far greater in men than in women, the ratio being 3 to 1.

Diagnosis

The diagnosis of a bleeding ulcer is not difficult. Slight oozing may be clinically unnoticed, but the least suspicion of bleeding renders a diagnosis simple. A chemical analysis of the gastric contents or feces for occult blood reveals the condition. When the loss of blood is 50 cc. or more the stool is tarry; in instances of great loss of blood melena or hematemesis may set in. The clinical symptoms of great loss of blood depend upon the rate at which the blood is lost. If gradual, the patient

can adjust himself to a very low percentage of hemoglobin and the blood volume can be adjusted physiologically. If, however, the loss of blood is rapid and there is no time for physiologic adjustment, symptoms of acute anemia are evident, i.e., rapid pulse, low blood pressure, dizziness and pallor.

DIFFERENTIAL DIAGNOSIS. Consideration should be given to the fact that bleeding from the gastrointestinal tract does not always imply lesions thereof. In most cases, the causes for bleeding lie in a pathologic process of the tract and are referred to as *intrinsic*. On the other hand, in some cases bleeding is due to some pathologic process outside the gastrointestinal tract, thus forming *extrinsic* sources.

In about 90 per cent of all cases bleeding is associated with peptic ulceration of the stomach or duodenum. Among other causative lesions are chronic gastritis, benign gastric tumor and gastrojejunal ulcer. Hemorrhage may also be caused by ulcers of Meckel's diverticulum, or by a neoplasm of the small bowel. Occasionally, bleeding arises from ulcerated esophageal varices, which are the result of obstruction to the flow of blood in the left gastric vein by cirrhosis of the liver, paraesophageal hernia, or some other cause. Hemorrhage from a peptic, gastric or duodenal, ulcer usually has a history of previous indigestion and of some kind of treatment for peptic ulcer. A valuable diagnostic point in bleeding gastric or duodenal peptic ulcer is that, in most cases, it is accompanied by pain, whereas bleeding from the stomach or duodenum from other causes is usually devoid of pain.

Benign tumors are more often painless and may be recognized roentgenologically and gastroscopically.

In *chronic gastritis* the bleeding is generally not accompanied by pain, and the condition can be recognized gastroscopically.

Splenic anemia produces recurrent hemorrhages but is not associated with pain. As the spleen becomes enlarged only in the late stage of the disease, the cause of bleeding may not be recognized but may be erroneously sought for in a gastric or duodenal ulceration. It is only later, when the spleen becomes enlarged, that the diagnosis becomes clear. In the earlier stages the physician has to depend chiefly upon the direct signs of gastric or duodenal ulcers.

Hepatic cirrhosis is frequently accompanied by an enlargement (hypertrophy) or diminution (atrophy) of the liver, ascites and icterus. In such cases the diagnosis is simple. If there is no change in the size of the liver and no icterus or ascites, the diagnosis may be difficult. In some cases an x-ray flat plate may show varices of the lower esophageal veins.

Diaphragmatic hernia may occasionally be the cause of gastric hemorrhage. The ring of the esophageal hiatus may compress the stomach and partially obstruct the flow of venous blood from the esophageal tributaries of the left gastric vein, and produce stasis and varices which may

rupture and result in hemorrhage. The diagnosis of diaphragmatic hernia can be established radiologically.

Ulceration or neoplasm of the small bowel are rare conditions which may run a course without any apparent symptoms. Therefore, the entire digestive tract should be x-rayed in obscure cases of gastrointestinal bleeding.

Bleeding from ulcerated Meckel's diverticulum is a rare occurrence. A diverticulum can be diagnosed radiologically, and if there is bleeding without evidence of gastric or duodenal disturbances, it may be presumed that the bleeding arises from the diverticulum.

Bleeding from ulcerated duodenal diverticulum is extremely rare. Nevertheless, the author had a patient with a bleeding diverticulum of the second portion of the duodenum.

Late postoperative hemorrhage occurs in some cases several months after a stomach operation. In the case of an operation for bleeding gastric or duodenal ulcer the recurrence may originate from the old ulcer. However, if the operation was performed for non-bleeding ulcer, the source of bleeding may be a peptic gastrojejunal ulcer at the stoma. This ulcer can be roentgenologically determined when it has penetrated not only the mucosa but also the muscular layer; however, if only the mucosa is involved the ulcer can be diagnosed only gastroscopically.

Among the **extrinsic** causes are malignant nephrosclerosis and hemorrhagic diathesis.

Treatment

The treatment of bleeding ulcer is medical or surgical. In the majority of cases medical treatment is the therapy of choice. It is chiefly attempted because the mortality rate is lower than in surgical intervention and only when medical treatment fails should surgery be resorted to. However, there are definite considerations which may necessitate immediate operation without preliminary medical treatment. It has been indubitably proved that the greatest majority of bleeding patients who succumb belong to the group above the age of forty-five. Ninety per cent of all fatal cases are patients over forty-five years, although only forty per cent of bleeding cases are above this age. This would mean that a patient below the age of forty-five may be reasonably expected to recover with medical treatment provided that blood pressure and pulse are checked every half hour or hour. If the blood pressure does not drop steadily and if the pulse is not accelerated, the patient has every chance of recovery. But, if the blood pressure drops below 90 mm. of Hg. and the pulse rises to about 120, immediate surgery is indicated. In cases in which the blood pressure falls to 70 mm. of Hg. and the pulse reaches 130, a blood transfusion must be given at once and followed immediately by operation.

In patients above forty-five years the requirements for procedure are

different: Since 90 per cent of all fatal cases, due to bleeding ulcer, belong to this age group, the surgeon should be prepared for surgical intervention, especially when the patients show symptoms of arteriosclerosis or hypertension, because in such cases there is no tendency for the bleeding to stop. Even if it does cease, it is apt to recur. If hemorrhage occurs in a patient with chronic ulcer of long standing, it may be assumed that there is an abundance of scar tissue which prevents retraction of the eroded end of the bleeding vessel so that there is no spontaneous cessation of the bleeding. It is evident that in such a case only surgery can stop the hemorrhage. If a large ulcer of the posterior gastric wall adheres to the pancreas and erodes a blood vessel, there is virtually no possibility that the bleeding will stop spontaneously. In such cases immediate surgery is indicated.

It is of special importance to determine whether the bleeding arises from a duodenal ulcer because it is attended by a higher mortality than hemorrhage from gastric ulcer. Moreover, a higher rate of mortality may be anticipated in men than in women. All these factors have to be borne in mind in the treatment of such patients.

MEDICAL TREATMENT. This consisted, until recent years, of rest in bed, morphine in sufficient doses to allay the patient's anxiety, abstinence from food for two or three days, to be followed by the intake of alkalizers and the gradual increase of the amount of bland food. At the same time, the acute anemia was checked by small doses of blood administered intravenously so as to increase the coagulability of the blood without raising the blood pressure. A large amount of blood should not be given because it may raise the blood pressure and dislodge thrombotic plugs in the bleeding vessels. At the present time essentially the same treatment is employed with the important exception of the dietary regimentation. We resort to blood transfusion only in cases of great loss of blood. We no longer rely upon the blood count and hemoglobin values to determine the amount of lost blood, because in the first few hours the blood count and percentage of hemoglobin may be almost normal despite considerable loss of blood. Usually, some hours elapse following hemorrhage during which the blood count as well as percentage of hemoglobin drop. This is due to dilution of blood by fluids coming from tissues of the body. At the present time, we rely more upon blood pressure and pulse rate. If the systolic pressure is below 90 Hg. and the pulse above 120, a blood transfusion of 250 cc. should be given.

DIETETIC CONSIDERATIONS. In 1906 Lenhartz pointed out the danger of abstinence from food as a part of medical treatment. He fed his patients with raw eggs and milk immediately following a hemorrhage and succeeded in reducing the rate of mortality among his patients to 2.14 per cent. However, Lenhartz' method did not attract the attention of the profession despite his great reputation.

In the United States, Andresen was the first to treat his patients by feeding rather than starving them. He based his treatment on the well known experiments conducted by the physiologist A. Carlson, who showed that an empty stomach manifests active peristalsis and secretion. Andresen claimed that it is more beneficial to the patient if the gastric secretion is diluted in bland food, thus diminishing the possibility of digesting the clots which plug the eroded vessel.

Despite excellent results reported by Andresen, the method was employed by only a few physicians until Meulengracht's report in 1933. He was able to reduce the mortality of cases to 1 per cent, his method being essentially the same as that employed by Lenhartz and Andresen. From the beginning Meulengracht gives the patient a liberal amount of food of high caloric and vitamin values and in purée form so as to avoid the additional burden of grinding upon the musculature of the stomach. This diet contains about 2,300 calories and is administered as follows: At 6.00 A.M., tea, white bread and butter; 9 A.M., oatmeal with milk, white bread and butter; 1.00 P.M., choice of meat balls, broiled chops, omelette, fish cakes, fish au gratin, puréed vegetables, vegetable soup, stewed apricots, apple sauce, rice or tapioca pudding; 3.00 P.M., cocoa; 6.00 P.M., white bread and butter, sliced meat, cheese and tea. In addition, the patient receives three times daily a powder containing: sodium bicarbonate, magnesium subcarbonate $\bar{a}\bar{a}$ 2.0, Extr. Hyosciamini 0.25. To combat anemia liver extract or iron should be given.

In 1937, Meulengracht published a report of 368 cases of serious bleeding from which only five succumbed to the disease (mortality 1.36 per cent). Since his publication this treatment has been widely used and numerous reports are published with a mortality rate of only 1.7 per cent.

In 1935, Woldman introduced a colloidal aluminum hydroxide drip method for the treatment of bleeding ulcers. Colloidal aluminum hydroxide is a creamy, white substance which is mildly astringent and does not irritate the gastric mucosa. The gastrointestinal tract does not absorb it; this substance neutralizes the acidity of the gastric juice and protects the ulcer by coating it. It is introduced into the stomach by means of an in-dwelling nasal Levine tube. Colloidal aluminum hydroxide has a marked viscosity and it is, therefore, necessary to use a siphonage apparatus. The apparatus employed by Woldman consists of an elevated flask filled with water and connected to a lowered empty bottle by means of a rubber tube. The empty bottle is connected with a syphon system which is filled with diluted colloidal aluminum hydroxide which, in turn, is connected with the in-dwelling nasal Levine tube. The water inflow releases the outflow of aluminum hydroxide at precisely the same rate, that is, drop by drop. Ten drops per minute is approximately the proper rate of flow. The bottles containing the water and aluminum hydroxide may be changed daily.

During the first twenty-four hours the patient takes 2 ounces of milk and cream every two hours. After the first day the diet includes cooked cereals, gelatine, custard, creamed soup, rice and tapioca pudding. The colloidal aluminum hydroxide is administered continuously, i.e., day and night for ten days.



FIG. 62. Excision of a gastric ulcer on the posterior stomach wall. A. Cutting of the gastrocolic ligament. Main picture. The posterior wall of the stomach is turned anteriorly. Ulcer is seen being adherent to the pancreas.

Several preparations are available containing aluminum hydroxide in tablet form, and are used in cases of bleeding peptic ulcer.

SURGICAL TREATMENT. Once surgery is decided upon, the patient must be properly prepared. If his blood pressure is below 90 mm. of Hg., a blood transfusion of about 250 cc. should be given; he also has to

be kept warm. The proper selection of anesthesia is an important consideration and, in most cases, local analgesia will suffice. If the blood pressure has not dropped below 110 mm. of Hg., spinal anesthesia may

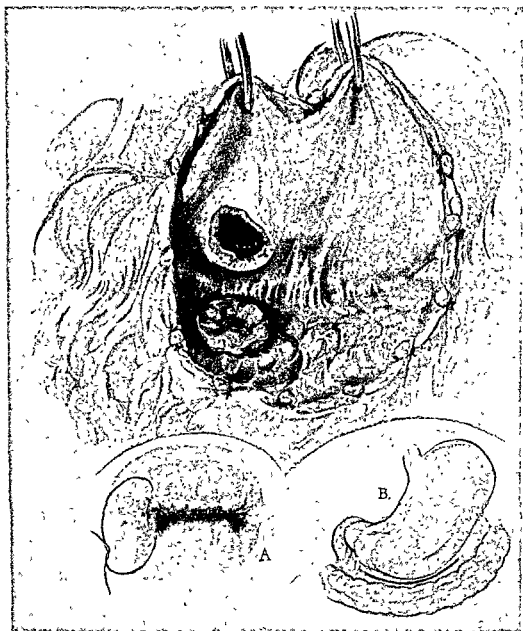


FIG. 63. Management of bleeding ulcer on the post. gastric wall and adherent to the pancreas. Ulcer excised from the stomach and left on the pancreas. A. Closure of the opening in the stomach. B. Position of the structures after operation. They are the same as before operation.

be employed. The choice and employment of the different types of anesthesia are described in the chapter on "Anesthesia in Urgent Surgery."

The operative procedure is determined by the site of the pathologic

process, i.e., whether it is in the stomach or duodenum, the condition of the patient and the extent of the ulcer.

In gastric ulcer the following operations may be performed: (1) If the patient is in a good general condition, subtotal gastrectomy is ad-

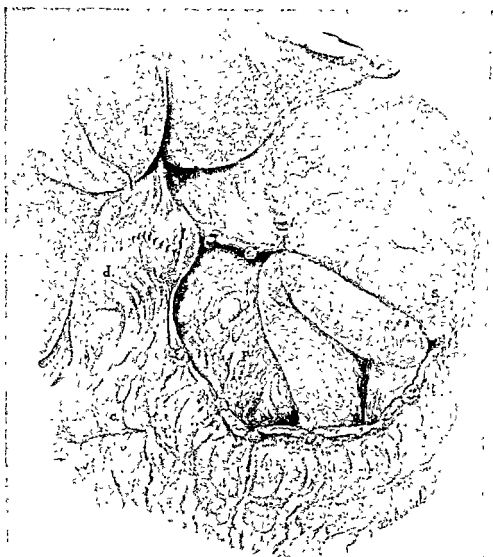


FIG. 64. Management of a bleeding duodenal ulcer by exposure and transfixion
Partial gastrectomy by Polya method (final view).

l—liver; d—duodenum; s—stomach; c—colon; p—pancreas.

visable. (2) If the patient's condition is not good, or if the ulcer is situated on the anterior wall, the ulcer is excised and the opening closed. In cases in which the ulcer is close to the pyloric outlet and closure of the stomach opening after excision of the ulcer would encroach upon the pyloric canal, this operation is supplemented by posterior gastro-

jejunostomy. If the ulcer is on the posterior gastric wall and adherent to the pancreas, the procedure is as follows:

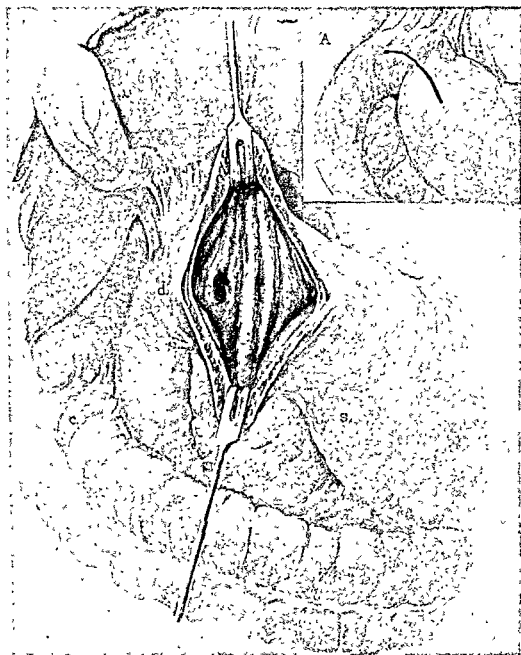


FIG. 65. Management of a bleeding ulcer on the posterior wall of duodenum. A. Incision through the anterior wall of pylorus and first portion of the duodenum. *Main picture.* The ulcer is exposed.

l—liver; d—duodenum; s—stomach; c—colon

First step: The gastrocolic ligament is divided for a distance of 3 to 4 inches so that the posterior gastric wall may be turned anteriorly (Fig. 62).

Second step: The posterior gastric wall is cut around the ulcer, leaving the latter adherent to the pancreas (Fig. 63).

Third step: The bleeders are cauterized or transfixed with silk ligatures.

Fourth step: The opening in the stomach is closed (Fig. 63, A).

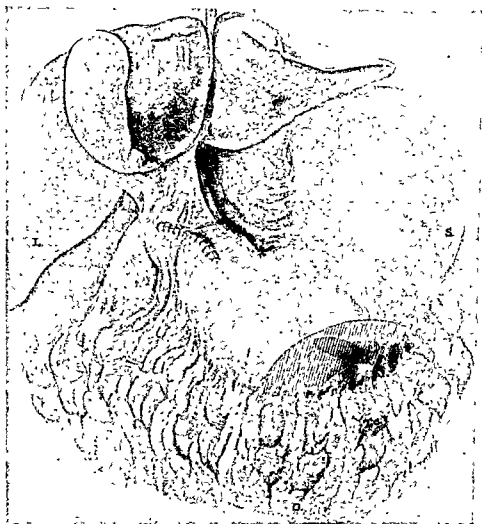


FIG 66. Management of duodenal bleeding ulcer (final view). T.C. Transverse colon
S. Stomach. O. Omentum. L. Liver.

Fifth step: The edges of the divided gastrocolic ligament are sutured to each other (Fig. 63, B).

If the ulcer lies on the lesser curvature, it is excised in a wedge-shaped form and, after closing the opening, posterior gastrojejunostomy is carried out.

The procedure in a bleeding *duodenal ulcer* is as follows: (1) If the

patient is in good condition the ulcer is exposed, the bleeders are cauterized or transfixed with silk ligatures, and this is followed by subtotal gastrectomy (Fig. 64). (2) If the patient's condition is not good, the technic is determined by the location of the ulcer, i.e., whether it is on the anterior or posterior duodenal wall. If it is on the former, the ulcer should be excised in a longitudinal direction and the duodenal opening closed transversely. If it is on the latter, the anterior wall of the duodenum is opened together with one or two inches of the anterior wall of the pylorus (Fig. 65). Suction is applied to give a clear view of the interior of the duodenum and bleeding ulcer; the latter is transfixed by means of a heavy silk ligature. The anterior duodenal and gastric walls are closed and a posterior gastrojejunostomy follows (Fig. 66).

Medical versus Surgical Treatment

It is generally agreed that surgical treatment has a considerably higher mortality rate than medical. The reasons for this are manifold: In the first place, those patients with bleeding ulcers which are medically treated frequently have moderate bleeding, whereas surgical cases represent profuse hemorrhage. Furthermore, most patients with bleeding ulcers are treated medically, and only when this treatment fails does the physician resort to operation. At that stage the patient is evidently in a serious condition.

In consideration of this factor, that medical treatment gives lower mortality than surgical, medical treatment is employed in individuals under the age of forty-five years and those who have not bled previously. In these cases the patient's blood pressure and pulse are checked every half hour. If the pulse does not increase and the blood pressure does not drop, medical treatment is continued. However, when the pulse reaches 120 and the blood pressure drops to 90 mm. of Hg., surgery is indicated. When the pulse reaches 130 and the blood pressure drops to 70, a blood transfusion is given without delay and is immediately followed by surgery.

In cases of repeated hemorrhages, and in patients above the age of forty-five, surgery should be considered from the onset. Hourly or semi-hourly recordings of blood pressure and pulse should be made; if the blood pressure drops steadily and the pulse increases rapidly, no time must be lost even if there is no melena or hematemesis.

Mortality

Twenty to 30 per cent of all patients with gastric or duodenal ulcer bleed, and of those who bleed about 5 per cent die from hemorrhage. Ninety per cent of the fatal cases are patients over forty-five, although only 40 per cent of hemorrhagic cases bleed after the age of forty-five. The mortality due to bleeding from duodenal ulcer is higher than that

caused by gastric ulcer. The incidence of hemorrhage is about three times more frequent in men than in women, and the ratio of mortality of men to women is still higher. In fact, women infrequently succumb to gastric or duodenal hemorrhage. Hemorrhage from chronic ulcer is more likely to be fatal than from an acute ulcer. The mortality rate rises rapidly with a second or third massive hemorrhage, closely following the initial bleeding. The mortality rate for patients operated upon depends greatly on the time which is allowed to elapse between hemorrhage and operation. Finsterer reported only 2.8 per cent mortality in early resections (twenty-four to forty-eight hours after hemorrhage), and 31 per cent in late resections. Most fatalities occur following the first hemorrhage. It is estimated that almost 75 per cent of all deaths occur after the first hemorrhage.

ACUTE DILATATION OF THE STOMACH

Acute dilatation of the stomach develops in 60 to 70 per cent of all cases as a postoperative complication. It may occur after any operation upon the abdomen, more frequently the gallbladder, but also after surgical intervention on any part of the body, such as genitalia, upper or lower extremities, and after childbirth. It is observed also after excessive intake of food or liquid and also in typhoid fever, pneumonia or diabetes. Acute dilatation of the stomach occasionally follows general or local anesthesia. It may appear a few hours after an operation, usually within twenty-four hours, but may take place some days later.

The causes of acute dilatation of the stomach are not known. Different theories have been propounded of which a neurogenic cause is the one most accepted. The dilated stomach may compress the duodenum directly by pressure on it, or by pushing the small bowel downward into the pelvic cavity; the descended bowels cause the mesentery and the superior mesenteric artery to become tense; the superior mesenteric artery squeezes the third portion of the duodenum against the spinal column, thus producing high duodenal obstruction. Owing to this obstruction, the gastric juice, bile and pancreatic juice have no outlet. The production of these juices may reach 1,000 cc. daily and the stomach becomes greatly distended. According to some reports, during distention the stomach occupied not only the abdominal cavity proper but also the pelvic cavity down to the symphysis pubis. In some cases on record the distention was so great that the serosa and the muscularis ruptured and mucosa became herniated.

SYMPTOMS. The early symptoms are not alike in different patients. An early symptom is that of a sense of discomfort. Inspection at that time reveals a moderate to pronounced distention of the abdomen in the epigastric region. Frequently the demarcation line between the costal arch and the abdominal wall disappears. By palpating the abdomen a succussion splash is often elicited. The pulse is accelerated, reaching up to 120. Nausea is a frequent early symptom. Vomiting is a late symptom and is not always present. If present, it is effortless; in other words, it is not a forceful ejection of fluid but rather an overflowing of the fluids from the mouth. Very often the patient has hiccoughs. The patient's temperature is subnormal (96° to 97° F.). Pain may be an early or late symptom; in some cases it does not appear at all. Blood pressure drops to shock level. Dehydration and oliguria are late symptoms.

X-RAY EXAMINATION. A flat plate will show a large homogenous mass reaching the pelvis or even the symphysis pubis. In case of doubt, 50 cc. of a diluted barium meal will clearly show the contours of the greater curvature and thus confirm the diagnosis.

The diagnosis is based on a greatly distended abdominal wall, accelerated pulse, low blood pressure, subnormal temperature, nausea, occasional vomiting and in doubtful cases on an x-ray examination.

TREATMENT. This consists of the following measures:

1. **Decompression of the stomach** by continuous suction through a catheter introduced into the stomach through the nose. The catheter should reach the greater curvature, as otherwise it may not reach the fluid level.

Decompression should be applied for at least thirty-six hours. The tube is removed only after the stomach has been tested for its mobile activity. This is done by occluding the rubber tubes leading from the stomach to the upper bottle and from that to the lower. The patient is given fluid by mouth. If he feels no discomfort after one hour's occlusion, the gastric tube may be withdrawn.

2. **Venoclysis** with normal saline or Hartmann's solution should be carried out at an average of 3,000 cc. in twenty-four hours. This is essential in order to replenish the fluid and mineral salts lost through suction from the stomach. Hartmann's solution replaces not only sodium chloride but also the potassium and calcium salts.

3. **Postural change of the patient.** In some cases emptying of the stomach is greatly enhanced by placing the patient either in a prone position (patient lying on the abdomen) or in the knee-chest position. The latter position was proposed by Julius Schnitzler of Vienna. We believe that suction correctly applied will adequately accomplish the decompression. Placing the patient in a knee-chest position after an abdominal operation taxes unnecessarily the patient's strength.

4. Among the **drugs** to contract the musculature of the stomach after the dilatation has been overcome by decompression, pitressin takes first place.

PERFORATION OF CARCINOMATOUS STOMACH

Perforation of a carcinomatous growth of the stomach is comparatively rare. Even in a large private practice it is very infrequently encountered and, for this reason, a study of this type of accident is only possible from histories recorded in large hospitals. It is exceedingly difficult to make a correct preoperative diagnosis so that the condition is, in many instances, mistaken for a perforated peptic ulcer. However, such a diagnostic error is, from a clinical viewpoint, not serious for the patient because immediate laparotomy is necessary in either condition and the true nature of the process is then recognized. In gastric perforation without palpable metastases, and in a patient of middle age who is losing weight and is anemic, it is advisable to carry out a biopsy before closing the opening. Sometimes, microscopic examination of a macroscopically appearing peptic ulcer may prove to be carcinoma and a radical operation at the earliest possible time is necessary.

Perforation of a gastric carcinoma was observed by Laennec in 1824. He not only described it correctly but was able to make a preoperative diagnosis to that effect.

As previously stated, perforation of gastric carcinoma is rare, occurring in 2 to 4 per cent of all cases of gastric carcinoma. It is observed more frequently in men than in women, the ratio being 3 to 1.

The clinical course of a perforated carcinomatous stomach does not run uniformly. It can be classified as (1) perforation into the general peritoneal cavity with a fulminating course, and (2) perforation with a silent clinical course.

FULMINATING PERFORATION. This group constitutes from 50 to 75 per cent of all cases of carcinomatous perforation. Its symptoms are identical with those found in perforated peptic ulcer. It differs from the peptic group only in that it occurs in persons at the ages of fifty to sixty whereas the peptic group is observed chiefly in individuals under the age of forty. There are occasional variations as to age and cases have been recorded in which perforation of gastric carcinomas occurred in the early twenties and in the eighties.

DIAGNOSIS. Diagnosis of gastric perforation as such is comparatively simple, and has been fully discussed in the chapter on "Acute Perforation of Peptic Ulcer." The difficulty arises only in the differentiation between carcinomatous and peptic ulceration. If the patient has been in the hospital before rupture took place, the diagnosis is not as difficult as when the patient is first seen after perforation. The data obtained during the patient's stay in the hospital before perforation took place, such as loss of weight, any quantitative changes in the free hydrochloric acid, x-ray of the stomach and so on, are invaluable. With all these data, a correct diagnosis can be made in about 50 per cent. But when the

patient is first seen after perforation, the board-like rigidity of the abdominal wall prevents palpation of a mass, even if it is present. The physician may resort to fluoroscopy to detect air bubbles in the free peritoneal cavity, and thus confirm a perforation, but he cannot employ barium to ascertain whether it is a case of ulcer or carcinoma. It is not advisable at this stage to aspirate the gastric contents for determination of its acidity. In other words, the most valuable information to facilitate a correct diagnosis is not available. Under these circumstances the physician has to rely upon the past history which may suggest carcinoma or peptic ulcer. It has to be borne in mind that the presence of free hydrochloric acid, even in an increased amount, does not exclude the possibility of gastric carcinoma. It has been found that in about 30 per cent of all cases of perforated gastric carcinoma, free hydrochloric acid was present; in some cases the amount was normal, in others it was increased in quantity.

A further factor that may mislead the surgeon is previous indigestion. It is commonly known that patients with a history of indigestion, dating back two or more years, suffer from peptic ulcer, and those with a shorter period of indigestion may have peptic ulcer or carcinoma. Generally, this is correct; however, it has to be remembered that 15 per cent of patients with perforated gastric carcinoma show in their history five years of indigestion; 35 per cent report indigestion for more than two years, and only 50 per cent state that indigestion started less than two years previous to perforation. It is believed that the reason for a long history of indigestion and free hydrochloric acid is that most cases of the fulminating type of perforation have carcinoma which developed at the site of a former ulcer.

Some of the erroneous diagnoses on record are: appendicitis, peritonitis, cholecystitis, internal hemorrhage and carcinoma of the stomach.

SILENT PERFORATION. This group comprises cases in which carcinoma appears in a formerly normal stomach and not at the site of previous peptic ulcer. No free hydrochloric acid is present and in about 90 per cent the history does not reveal previous indigestion of long duration. The perforation sets in gradually, and protective adhesions are formed so that when an actual perforation occurs the escaped contents do not spread over the peritoneal cavity but, in many cases, become encapsulated.

Ninety per cent of all patients reveal a history of indigestion of less than one year's duration. When perforation occurs some signs of the accident become apparent, such as sudden pallor, anxiety of expression and rise in the pulse rate. However, there is no severe pain or marked tenderness. A correct diagnosis of these cases is extremely difficult. Statistical surveys show that erroneous diagnoses in this group reach up to 90 per cent, while in fulminating perforation errors are made in about

50 per cent of the cases. Tuberculous peritonitis, malignant peritonitis and internal hemorrhage have been mistakenly diagnosed for silent perforation.

The *site of perforation* is, in the order of frequency, on the lesser curvature in the prepyloric region, on the anterior wall of the prepyloric region, on the cardiac end of the lesser curvature, and on the greater curvature. It is rare on the fundus, the anterior wall of the body, and on the posterior wall of the stomach.

PATHOLOGY. The most frequently encountered diseases are adenocarcinoma, ulcerative carcinoma and medullary carcinoma. The extent of the perforation is usually less than one-quarter inch in diameter although in about 15 per cent of the recorded cases it was larger than a one-cent piece.

Metastases are present in about 50 per cent. They are most frequently found in the omentum and less often in the liver and pancreas. The frequency of metastatic findings illustrates that perforation occurs rather late in the course of gastric carcinoma.

Upon analyzing the symptoms and site of location in fulminating and silent perforations, it is apparent that the first group shows a history of indigestion extending for several years, free acidity, and location along the lesser curvature in the prepyloric region. In other words, fulminating perforation occurs in carcinomas which are found where ordinarily peptic ulcer is located. Therefore, ulcers which are transformed into carcinoma have a tendency to become fulminating after perforation, while the silent type is observed in gastric carcinomas which develop independently of any other lesion.

COMPLICATION OF A PERFORATED CARCINOMATOUS ULCER. The most frequent complication of a perforated carcinomatous ulcer is contamination of the general peritoneal cavity with ensuing peritonitis. Next in frequency is the formation of a perigastric abscess, gastrocolic fistula, subdiaphragmatic abscess, perforation into the abdominal wall, gastrocholedochal fistula, perforation into the pleural cavity, perforation into the spleen, and gastrojejunal fistula.

TREATMENT. The treatment is essentially surgical. Operation may be performed immediately upon recognizing the condition, or some hours later if the patient is in shock. A blood transfusion is always advisable as a preliminary step in carcinomatous patients. The type of operation to be carried out is decided upon when the abdomen has been opened. Several procedures are available: simple closure, excision with closure, closure with gastroenterostomy, and gastrectomy. The ideal operation is gastrectomy but it is not always possible owing to its high mortality rate. The simplest procedure is closure. However, owing to the friability of the edges, there is frequently leakage and death from peritonitis. Aird reported a series of twenty-three cases of perforated carcinomatous ulcer

treated by simple closure; only three patients lived long enough to leave the hospital. This record demonstrates that the mortality for this type of treatment is 85 per cent. In some cases, simple closure may be contra-indicated, e.g., in a case in which the perforation lies close to the duodenum so that closure may produce narrowing of the pyloric canal. In such instances closure must be supplemented by gastroenterostomy. Sometimes it is possible to excise the growth and close the opening. Special care has to be taken not to constrict the lumen of the stomach. It is unquestionable that in perforation without metastases, with the patient in a good general condition, gastrectomy is the operation of choice. However, if the patient is not in a good condition, closure or excision with closure is an emergency measure, and gastrectomy may be performed ten to twelve days later when the patient has improved.

MORTALITY. The mortality rate for all types of operation is extremely high. About 60 per cent succumb within the first ten days following operation and a further 20 per cent within the next six weeks.

PHLEGMONOUS GASTRITIS

Although phlegmonous gastritis is rare, the fact that more than 300 cases have been published justifies calling the attention of the surgeon to this disease. The disease begins as a localized cellulitis of the submucous layer of the stomach and later becomes diffused or circumscribed, the first type being more frequent.

ETIOLOGY. Different pyogenic micro-organisms may produce this condition. Sundberg states that in ninety-five cases of phlegmonous gastritis, which were examined bacteriologically, the streptococcus was found in seventy-one cases. In the rest of the cases, staphylococci, pneumococci and bacillus coli were found.

The method of entrance into the stomach is through the blood stream from other diseased organs, such as infected tonsils, gums, lungs or through the mucosa of the stomach injured by lesions such as peptic ulcer or carcinoma.

Whichever the mode of entrance, the micro-organisms grow faster in the case of hypoacidity of the gastric contents. Many investigators have found that in most cases the hydrochloric acid is greatly diminished. Stieda reported that in sixty-four gastric operations performed on patients with low hydrochloric values, the incidence of postoperative infection was 30 per cent, while in thirty-five cases with high acid values, it was only 2.8 per cent.

INCIDENCE. This is not as rare a condition as it was assumed in the past. Riegel found only fifty cases in the literature up to 1905; Bumm, in 1925, collected as many as 219 cases, and at this time the number exceeds 300. Lawrence recorded only two cases from 5,000 autopsies.

AGE. Phlegmonous gastritis may occur at any age, but is usually encountered between the ages of thirty and sixty. According to Eliason and Murray-Wright, about 83 per cent of all cases are observed in this age period.

SEX. It is predominant in the male at a ratio of 3 to 1.

PATHOLOGY. The process usually affects the submucous layer and is, as a rule, confined to this area, although in some patients it involves the muscularis and serosa, in which case free exudate may be found in the free peritoneal cavity. The mucosa is rarely affected. The inflammation is either **diffuse**, involving the entire stomach from the cardiac to the pyloric end, or **circumscribed**, affecting merely a portion of the stomach close to the pylorus. In the diffuse form both walls are usually involved, whereas the circumscribed type attacks only the anterior wall. The process seldom extends beyond the limits of the pylorus or the cardia spreading to the duodenum and lower portion of the esophagus. In circumscribed phlegmonous gastritis the affected part is pale, edematous, and hard on palpation; it may cause marked bulging of the sero-

muscular or mucosa layers; occasionally, the seromuscular layer is ruptured.

In diffuse phlegmonous gastritis the entire wall of the stomach is edematous, and the submucous layer, instead of being a thin sheet, becomes very thick and may reach two-thirds of the entire thickness of the gastric wall. A purulent exudate can be squeezed out from the cut gastric wall. The rugae of the mucosa are absent and the latter presents a flat surface. The diffuse type occurs more frequently than the localized.

Microscopically, the submucous layer is thick and edematous and considerably infiltrated by polymorphonuclear leukocytes. In some places the submucous layer shows areas of necrosis. The adjacent lymph nodes reveal marked inflammatory changes and infiltration with polymorphonuclear leukocytes.

SYMPTOMS. The diffuse type differs from the circumscribed in that it is more acute but symptoms of pyloric obstruction are less prominent. Phlegmonous gastritis presents a picture of what is commonly termed "acute surgical abdomen" and septicemia. The onset is sudden with intense and constant pain localized in the epigastric region. The pain diminishes if the patient changes from a horizontal to a sitting position, a sign of great diagnostic significance. The pain is followed by vomiting; occasionally, the vomitus contains pus which is also of diagnostic significance. The epigastric region is tender and rigid. The abdominal wall may be protuberant in the epigastric region and flaccid in its lower half. The temperature ranges from 104 to 106°F. but may drop to subnormal values in case of prostration. The tongue is dry and coated. Headache, malaise and thirst are early symptoms.

Examination of the gastric contents shows, in the majority of cases, a marked hypo-acidity. The leukocyte count ranges from 15,000 to 30,000 per cubic millimeter.

A characteristic feature of this condition is that pain, vomiting and fever persist throughout the entire course of the disease, which varies from four days to two weeks.

In the circumscribed form the symptoms are not as acute, but as the process is close to the pylorus, symptoms of pyloric obstruction are frequently observed. For this reason an erroneous diagnosis of carcinoma of the pylorus has been made in a number of cases.

The differential diagnosis must exclude conditions which run a septic course or those producing an acute abdominal disease. One has to rule out pneumonia, acute pancreatitis, perforated gastric or duodenal ulcer and acute cholecystitis. That the diagnosis is extremely difficult is shown by the fact that a correct preoperative diagnosis has been made only in a very few instances, and that in many instances the condition has not been recognized even during operation, so that its true nature

was ascertained only on autopsy. Most frequently the preoperative diagnosis has been perforated peptic ulcer or acute pancreatitis. In differentiating phlegmonous gastritis from a perforated peptic ulcer it must

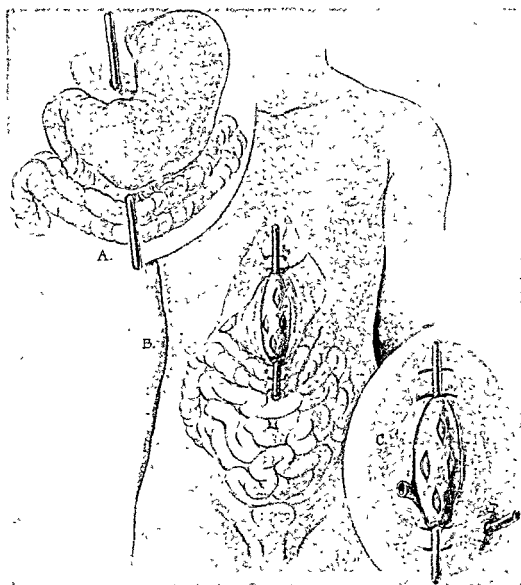


FIG. 67. Management of phlegmonous gastritis. A. Marsupialization of the stomach. B. Seromuscular incisions. C. Final view with two drains inserted.

be borne in mind that in the former pain diminishes when the patient changes from the horizontal to the vertical position, while in the latter the pain increases when the patient makes any movement.

TREATMENT. The mortality rate, whether the treatment be medical or surgical, reaches as high as 95 per cent. Nevertheless, surgery offers

some hope provided operation is done early. It is always advisable to give the patient fluids parenterally and sulfa derivatives and penicillin prior to and for some time after the operation.

The type of surgery to be employed depends upon the condition of the patient and upon the extent of the involved area of the stomach. If the condition of the patient is fair, partial gastrectomy may be carried out if the phlegmon is circumscribed, while subtotal or even a total gastrectomy is indicated if the process is diffuse.

If the condition of the patient is bad, Weirstein and Klein have advised marsupialization of the diseased part provided that only the anterior wall is affected, and extraperitonization if both walls are involved. When this is accomplished, several small seromuscular incisions of the gastric walls are made in order to drain the submucosal space. The operation is completed by a temporary jejunostomy which enables the feeding of the patient.

The extraperitonization is by no means a simple procedure, because the cardiac end is rather immobile and, in diffuse cases, the entire stomach is affected so that the entire stomach should be extraperitonized. That is the reason why some surgeons advise making multiple punctures in a diffuse phlegmon without extraperitonization, resorting to marsupialization or extraperitonization only in the circumscribed phlegmon.

The technic of extraperitonization is as follows: A small opening is made in the gastrohepatic and gastrocolic ligaments; the affected portion of the stomach is delivered outside the peritoneal cavity; a glass rod is carried through both openings behind the posterior gastric wall and rests on the abdominal wall; the edges of the peritoneum are sutured to the stomach, thus extraperitonizing its protruded portion (Fig. 67). Several small seromuscular incisions are made on the anterior gastric wall.

THE PROGNOSIS is very grave, irrespective of the type of treatment employed.

FOREIGN BODIES IN THE STOMACH

Foreign bodies in the stomach do not belong to the province of urgent surgery as long as no complications arise. They may remain in the stomach for years, even decades, without showing any symptoms. However, from time to time they produce complications, such as ulcerations, perforations and obstruction of the stomach or bowel, in which case they become problems of urgent surgery. Depending upon the substance of the foreign bodies they may be classified into several groups:

1. **FOREIGN BODIES** such as nails, pins, tacks and other objects, which carpenters, upholsterers and others have a habit of holding in their mouths while working. Professional swallows may have a great number of different foreign bodies in their stomachs (Fig. 68).

2. **TRICHOBEZOARS** consist of hairballs of different size and weight. They are usually single, though in some cases they may be multiple. If multiple, their sizes may be small, while a single one may occupy the entire lumen of the stomach, reaching as much as six and a half pounds in weight. They may be of different shape, e.g., U-shaped, bean-shaped, oval and the like (Fig. 69).

Irrespective of the color of the hair ingested, the trichobezoars are usually dark brown or black. This is true even if the swallowed hair was blonde or red, which evidently is due to a reaction of the gastric contents on the hair.

3. **PHYTOBEZOARS** are composed of material of vegetable origin, such as fibers, seeds, leaves, the skin of fruits, and the like. They mold together and form a mass. The fruit most frequently causing such a formation is persimmon. They are brittle and, in cross section, homogenous gummy material can be observed interspersed with skins of fruits or seeds. Other fruits or vegetables that may produce phytobezoars are coconut fibers, celery fibers, skins of grapes, prunes and others (Fig. 70).

4. **CONCRETIONS** may be formed by accumulated drugs, such as salol or bismuth. However, they are generally produced by addicts drinking furniture polish who crave its high alcoholic content. When such an addict drinks water to quench the thirst the resin contained in the polish precipitates and forms into a mold like that of the stomach.

ETIOLOGY. Trichobezoars usually occur in young individuals, and about 80 per cent of all cases occur between the ages of twenty and thirty, while phytobezoars are observed in persons past fifty.

SEX. Trichobezoars occur in 90 per cent of women and 10 per cent of men. Phytobezoars are found in 25 per cent of women and 75 per cent of men.

SYMPTOMS. The symptoms vary depending upon the degree of gastric irritation, the presence or absence of associated pathologic processes and their character. If ulceration of the gastric wall is present, the symptoms

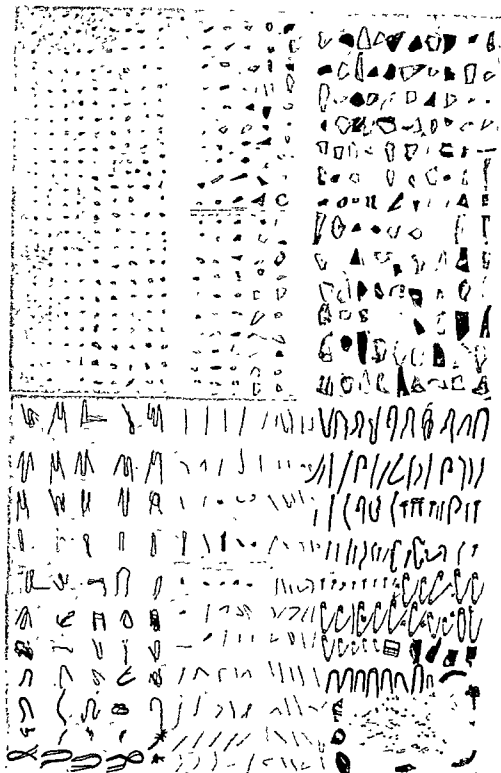


FIG. 68A, B. Foreign bodies in the stomach (courtesy of the *New England Journal of Medicine* and Doctor Philip H. Wheeler).

differ from those in which obstruction is the main complication. However, if there are no complications, the presence of trichobezoars may not give rise to symptoms for several years and even decades. When symptoms do arise, they are mild at first; there may be anorexia, dyspepsia, some loss of weight, feeling of heaviness in the epigastric region, and a foul odor from the mouth which is characteristic of bezoars. Later, the patient may have attacks of nausea, vomiting, epigastric pain, constipation or diarrhea. A palpable mass can be located in the epigastric region in about 90 per cent of all cases. With phytobezoars the symptom-free period is rather short; the history reveals ingestion of a great number of persimmons. Some days later there is nausea, vomiting and cramping pain; there may be slight fever. After a few more days the attack subsides and for a few weeks the patient is comfortable. These attacks recur every few weeks and persist for a few days. A palpable mass in the epigastric region can be felt in about 60 per cent of the patients.



FIG. 69. Trichobezoars in the stomach (courtesy of *Surgery* and Drs. A. Ochsner and M. De Bakey).

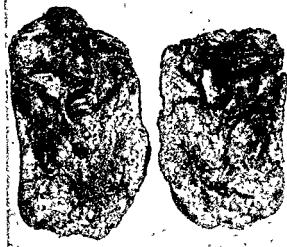


FIG. 70. Phytobezoars in the stomach (courtesy of *Surgery* and Drs. Alton Ochsner and M. De Bakey).

Roentgenographically bezoars can be diagnosed in about 70 per cent of all

cases. Metallic foreign bodies are recognized in 100 per cent.

The diagnosis may be comparatively simple or very difficult, depending upon the complications and to what extent they mask the original cause underlying the disease. The presence of a large movable epigastric mass, accompanied by pain, nausea, vomiting, absence of fever, the characteristic picture of bismuth spreading under fluoroscopy, facilitate the diagnosis which becomes almost a certainty of gastroscopy.

THE TREATMENT is surgical. It consists of gastrotomy, removal of the bezoar and closing of the stomach. It should be remembered that the contents of the stomach are extremely foul and, therefore, before opening the stomach the affected portion of the gastric wall should be walled off from the rest of the peritoneal cavity to prevent contamination. It should also be borne in mind that trichobezoars occasionally extend into the duodenum; therefore, the duodenum should be examined for their presence before the abdomen is closed.

PROGNOSIS. The mortality rate among patients not operated upon is very high, for trichobezoars 72.2 per cent and for phytobezoars 55 per cent. A fatal outcome is mainly caused by such complications as obstruction and perforation. The operative mortality is comparatively low, being 4.8 per cent for trichobezoars and 7 per cent for phytobezoars.

GASTRIC DIVERTICULA

Diverticula of the stomach ordinarily do not fall within the scope of emergency or urgent surgery, because they do not cause complications produced by diverticula of other parts of the gastrointestinal tract, such as peritonitis, gangrene, acute diverticulitis and perforation. However, they may be mistaken for lesions which attack the stomach more frequently, such as perforating gastric ulcer or carcinoma and, for this reason, they should be discussed at least briefly.

INCIDENCE. Diverticula are found very rarely; they trail the diverticula of other portions of the gastrointestinal tract. In the order of frequency they are located in the colon, duodenum, esophagus, stomach and in the small bowel. Gastric diverticula are observed in about 0.1 per cent in necropsies; on roentgenologic examinations they are found once in about 4,000 cases.

AGE. Diverticula occur most frequently in middle-aged persons although cases have been recorded in which this condition occurred in an infant four months old and in a child aged seven. Diverticula are found about as frequently in men as in women.

PATHOLOGY. Gastric diverticula are either of the true or false variety (Fig. 71). They are situated most frequently in the posterior gastric wall, close to the lesser curvature and the cardia, so that they are not easily accessible for operative intervention and can be overlooked in exploratory laparotomies. However, diverticula may be found on the anterior wall of the antrum, on the greater curvature and in any other part of the stomach. They vary from 0.1 to 5 cm. in diameter and usually have a narrow neck. The orifice leading into the diverticulum has the appearance of a circular hole; the margins are sharply defined, rounded and smooth.

The mucosa of the adjoining parts of the stomach is normal. This appearance, which can be demonstrated gastroscopically, is in marked contrast to that of a penetrating gastric ulcer, the base of which is usually covered with a gray or white exudate, with the adjoining gastric mucosa showing signs of inflammation. A penetrating carcinoma will produce symptoms of ulceration at the base of the defect and the surrounding margins are irregular and infiltrated.

SYMPTOMS. In about two-thirds of all cases of diverticulum of the stomach the disease is not revealed by any symptoms, but is found accidentally while operating for some other condition, or is discovered in an x-ray examination. Symptoms may become apparent when there is a narrow sac from which food entering the diverticulum cannot be eliminated, thus stagnating and producing inflammation.

The symptoms may be as follows: (1) **Pain**, described as dull or burning, in the upper abdomen, behind the xyphoid or under the left costal

margin. Food may aggravate the pain or relieve it, or may have no bearing on its intensity. (2) Vomiting. (3) Epigastric tenderness is circumscribed and mild or moderate. (4) Bleeding may occur, ranging from



FIG. 71. Gastric diverticula. A. True. B. False. C. Traction diverticulum

mild oozing to a profuse hemorrhage. Associated lesions are present in about 35 per cent of all cases of gastric diverticula, such as benign tumors, malignant tumors and gastric ulcers. These lesions may cause symptoms which are characteristic for each particular type. When a

gastric diverticulum is not associated with any other pathologic process, it usually runs a symptom-free course, in contrast to diverticula of the large bowel which may give rise to such complications as peritonitis, gangrene, perforation of its wall, acute suppurative diverticulitis, or local abscess.

DIAGNOSIS. The diagnosis is made roentgenologically or gastroscopically. A roentgenologic examination may not reveal a diverticulum if the neck is too narrow for the contrast medium to enter the sac. Expert gastroscopy may detect the condition in the majority of cases. It can be differentiated from penetrating gastric ulcer or gastric carcinoma by studying the edges of the opening and the character of the adjoining gastric mucosa. The different appearance of these three conditions was discussed at the outset of this chapter in a passage on "Pathology."

TREATMENT. When the condition does not reveal any symptoms and is found accidentally, it requires no treatment. However, if the disease is manifested by symptoms, the treatment may be medical or surgical. The medical treatment consists of "postural drainage." The patient drinks some water and then assumes a definite position, i.e., he may lie first on his right side, then turn onto the left side or the abdomen. Some surgeons claim that they can prove roentgenologically that such posture produces drainage; they were able to show that barium lodged in the diverticulum disappears after the intake of water. If medical treatment proves ineffective and the symptoms become more noticeable, operation should be carried out. However, it should be emphasized here again that the indications for surgery of gastric diverticula are rare because they seldom cause complications. Nevertheless, some surgeons claim that diverticula may become malignant.

If the diverticulum is situated on the anterior wall of the antrum, the exposure is simple; but when located on the posterior wall, the exposure is more complicated. The gastrocolic ligament is severed, the posterior wall turned anteriorly, and the diverticulum is removed. The stomach wall is closed in two or three layers. If the diverticulum is close to the cardia on the posterior gastric wall and close to the lesser curvature, the gastrocolic ligament is cut along its entire length and the stomach is turned from left to right along the lesser curvature as an axis so that the diverticulum is exposed and removed.

VOLVULUS OF THE STOMACH

Volvulus of the stomach is a rare condition. It was first described by Berti in 1866 and, according to Weeder, only seventy cases were reported in the literature up to 1935. Since then the total number of cases has reached about eighty.

Morbid Anatomy

Volvulus may be total or partial. In the first group the entire stomach is involved in the rotation, while in the second group only a portion of the stomach is affected. In complete volvulus the organ may be rolled around a line which connects the cardia with the pylorus, the cardiopyloric line, thus producing a true volvulus; or it may be twisted around a line which connects the middle of the lesser with the middle of the greater curvatures, thus producing torsion of the stomach and not a true volvulus. However, most investigators apply the term "volvulus" to either condition. The first group, i.e., that in which the volvulus occurs around the cardiopyloric line is known as *volvulus organo-axialis*; torsion along the line connecting both curvatures is termed *volvulus mesenterio-axialis*. The majority of recorded cases are of the latter type.

VOLVULUS MESENTERIO-AXIALIS. In this type the rotation takes place around a line connecting the middle of the lesser with the middle of the greater curvature. (Fig. 72, A) Most of the rotation is made by the pyloric portion. The latter becomes considerably dilated, due to occlusion of the pylorus produced by the twisting and kinking which results from its change of position. The distended pyloric pouch crosses the fundus and lies with its posterior surface facing forward. In cases of pronounced torsion it is not only the pyloric but also the cardiac orifices of the stomach which become occluded.

The right half of the transverse colon is carried with the pylorus upward and to the left, whence it descends sharply over the distended pyloric pouch under and behind it, reappearing again on the left of the splenic flexure. The sharp kink in the right half of the transverse colon produced by its displacement causes marked distention of the right half of the colon.

VOLVULUS ORGANO-AXIALIS. This is produced by rotation of the stomach along the cardiopyloric line. It is a true type of volvulus. According to the course of the greater curvature in the process of rotation, the organo-axialis type has two varieties: (1) *Volvulus anterior infracolicus*, (Fig. 73) in which the greater curvature rolls along the anterior abdominal wall upon the anterior surface of the stomach. While rolling upward, the greater curvature draws the transverse colon above the stomach. (2) *Volvulus posterior infracolicus* (Fig. 74) is the variety in which the greater curvature rolls along the posterior abdominal wall upon the posterior gastric wall upward and to the right. It carries with

it the gastrocolic ligament and the transverse colon so that, also in this type, the colon lies above the lesser curvature.

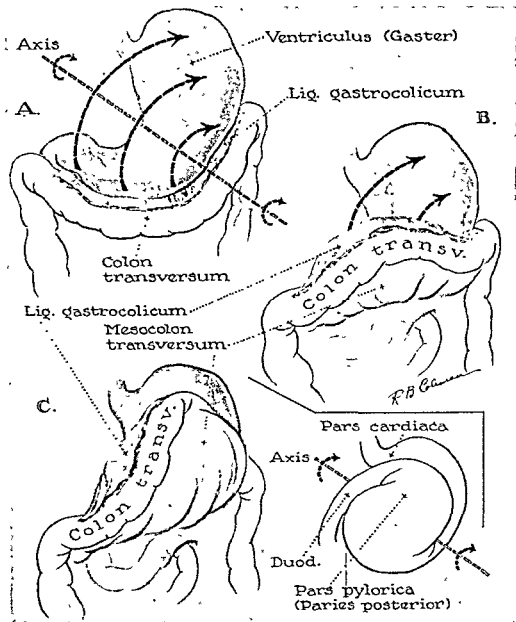


FIG. 72. Volvulus mesenterio-axialis. A. Normal position of stomach with arrows indicating direction of rotation around axis. B. Figure showing rotation begun and transverse colon being pulled upwards with greater curvature of stomach. C. Completed volvulus. Diagram shows position of lower half of stomach at this stage.

In the anterior infracolic group the stomach is veiled by the gastrocolic ligament. In some cases of volvulus organo-axialis the transverse colon does not follow the greater curvature; this is possible only when the gastrocolic ligament is very long or when this ligament has ruptured.

Partial Volvulus. This is always of the organo-axial type. In this variety the pyloric part of the greater curvature rotates more freely

than the fundal part; the latter may even remain unchanged. Thus, the pyloric portion becomes separated from the cardiac by a V-shaped sulcus on the lesser curvature. The rotation in partial volvulus never exceeds 180° .

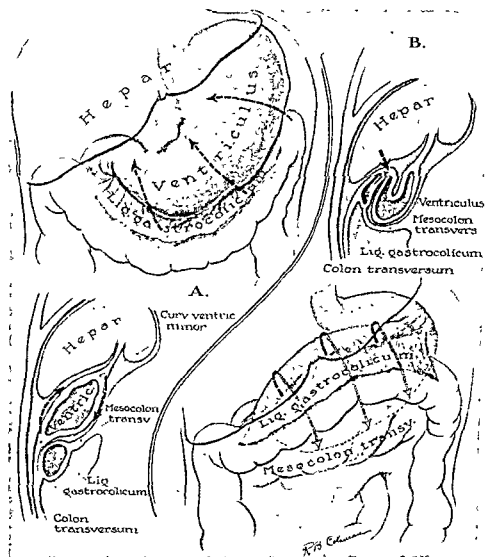


FIG. 73. Volvulus organo-axialis anterior infracolic. A. Stomach in normal position. Arrows indicate direction of rotation. B. Stomach rotated on cardiopyloric line bringing greater curvature up under liver, and transverse colon pulled up above level of lesser curvature of stomach. Arrow in median section indicates position of greater curvature. Arrows in frontal view show rotation which has taken place.

Partial volvulus is always associated with an intra- or extragastric lesion, such as hour-glass stomach, gastric tumor, perigastritis, diaphragmatic hernia, tears in the transverse mesocolon, tears in the gastro-hepatic ligament, and the like.

Total Volvulus. In total volvulus patients often have a marked degree

of gastropptosis; in other words, it is more liable to occur where elongated ligaments are connected with the stomach which produce a marked mobility of the pylorus sufficient to come in close proximity to the cardia. If the pylorus crosses the cardia ("Crossing of the pedicles"), a volvulus takes place.

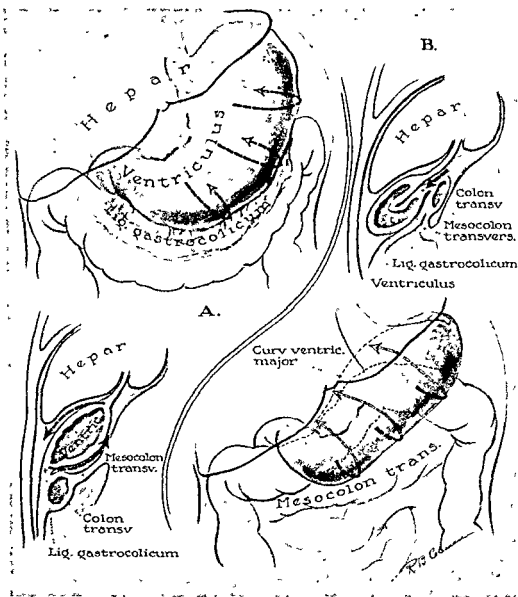


FIG. 74. Volvulus organo-axialis posterioris infracolic. A. Stomach in normal position. Arrows indicate direction of rotation of stomach in this type of volvulus. B. Stomach rotated on cardio pyloric line bringing greater curvature posterior and under liver. Transverse colon pulled up posteriorly as greater curvature rotates and transverse colon comes to lie above level of lesser curvature of stomach. Arrow in midsagittal section indicates position of greater curvature. Arrows in frontal view show rotation which has taken place.

ETIOLOGY. Volvulus of the stomach is a rare disease. It occurs mostly in middle-aged persons though cases of volvulus have been reported occurring in an infant ten months old and in a child of two years.

SYMPTOMS. The condition may set in suddenly or insidiously. The symptoms may vary according to the degree of the volvulus. If it does not close the stomach completely, it manifests itself by pain in the left hypochondrium or in the epigastrium. It is followed by vomiting. The abdomen becomes distended in the epigastric region and flaccid in the rest of the abdomen. Upon palpation the distended part gives the impression of an elastic balloon which may be slightly rigid. The roentgenogram shows a high position of the left half of the diaphragm and an air-filled space with a line of fluid. Later when the volvulus is complete, the symptoms become more aggravating. The pain increases; respiration becomes frequent and shallow; the face is livid; the epigastric region is markedly distended in contrast to the rest of the abdomen; the pulse is rapid and small; and the patient prefers a sitting position. It is characteristic that vomiting ceases but retching prevails. The patient is dehydrated and thirsty but as soon as he takes water or any liquid he regurgitates it immediately although spontaneous vomiting ceases. This latter symptom is pathognomonic for occlusion of the cardia, irrespective of the cause. It is also significant that it is impossible to introduce a gastric tube. However, this diagnostic method should be cautiously applied because there may be trophic changes in the wall of the twisted cardia and an attempt to introduce a tube forcibly may lead to perforation.

DIAGNOSIS. A correct preoperative diagnosis is by no means easy and, therefore, is not frequently made. Nevertheless, we believe that in most instances this is due to inadequate clinical experience. If the physician would consider volvulus and bear in mind that the patient vomits only in the early stage of this condition while retching continues, and that the intake of fluids is immediately followed by regurgitation, the diagnosis would be less difficult.

The differential diagnosis should exclude the following conditions: (1) **Acute dilatation of the stomach** in which vomitus does not cease. The vomitus contains bile and the introduction of a gastric tube meets with no obstruction. (2) **In high intestinal obstruction** the vomited matter contains bile and later has a fecal odor. The swelling is not circumscribed, the tympanic pitch is not as high; swallowing of water is not followed by immediate eructation. (3) **In acute pancreatitis** the pain is more severe and radiates to the back; there is no retching or immediate eructation of swallowed water. Distention of the abdomen is moderate. (4) **Perforated viscus** shows an acute or even hyperacute onset, marked rigidity and tenderness; distention is not noticeable unless seen at a later stage when symptoms of peritonitis appear. (5) **Acute mesenteric thrombosis**; severe vomiting; distention is moderate and uniform; frequently a heart lesion is present.

THERAPY. Only surgical treatment should be employed. If no treatment is given all patients invariably die. Operation for total volvulus results in recovery in about 60 per cent, and for partial volvulus in almost 100 per cent of all cases.

TECHNIC. The abdomen is opened by a right paramedian incision extending from the xyphoid process down to the level of the umbilicus. Exposure and exploration of the abdomen should be done carefully, because the gastrocolic ligament often lies in front of the stomach and its vessels may be injured by rough manipulation. As soon as the diagnosis is made it is advisable to puncture the stomach with a trocar, after having taken the precaution to prevent the escape of gastric contents into the free peritoneal cavity by walling off the place where the trocar is to be introduced. When the contents are aspirated, it is advisable immediately to close the opening in the stomach and not to defer closure until detorsion of the stomach has been attained, because the open part of the gastric wall may retract into the abdominal cavity and it may be difficult to locate it later. Cases have been reported in which the surgeon attempted to bring the distended stomach outside the abdominal cavity with the result that the stomach ruptured. In another recorded case the manipulation was followed by vomiting, with death ensuing from suffocation. The danger of attempting to bring the overdistended stomach outside the abdominal cavity may also involve rupture of the spleen or of the splenic vessels situated in the gastrosplenic ligament. There is also the risk of rupturing the overdistended loop of the transverse colon which often lies above the stomach and is caught between the stomach and the diaphragm.

When the stomach is deflated, it is placed in its normal position and should be examined for diaphragmatic hernia. If this is absent, the abdomen is closed but in a case of hernia an attempt must be made to *reduce it. The hernia should be repaired at once if the condition of the patient permits it or later if the condition is bad.* In a case of irreducible hernia the surgeon has no way of ascertaining whether it is merely irreducible or incarcerated, and he has no choice but to proceed to free the sac from its contents.

In order to prevent recurrence, some surgeons perform gastropexy, others gastroenterostomy; their effectiveness is questionable.

In case the stomach wall is found to be gangrenous, the affected portions have to be removed, always bearing in mind to keep manipulations at a minimum.

When the operation is completed it is advisable to employ continuous decompression of the stomach by means of a nasal catheter left in place for three days. This enables the overdistended gastric wall to resume its motor strength and prevent it from becoming distended again.

INJURIES AND WOUNDS OF THE STOMACH

The stomach may be injured by an extraneous force in several ways: In one type, the external force penetrates the walls surrounding the abdominal cavity; this may be the anterior abdominal wall, the chest wall and the diaphragm, or the lumbar region. To this category belong gunshot and stab wounds. Another type is the so-called subcutaneous rupture in which the abdominal wall remains intact but a wound is inflicted upon the stomach by transmission of the blow through the abdominal wall. To this group belong cases in which the abdomen has been kicked, run over, or pressed against a hard and immobile object.

There is a third type of injury which is of great significance because it exacts the surgeon's skill and judgment more than the first two types, namely, injuries to the stomach resulting from the intake of corrosive substances. Each of the three types has its characteristic features, not only from the etiologic viewpoint but also from that of a difference in the symptoms, method of treatment and prognosis.

Direct Injury to the Stomach

Direct injury is caused principally by gunshot wounds or penetrating instruments such as a knife, dagger, arrow, lance or other object.

The wound produced in the gastric wall may be small or large; it may be single or multiple; it may injure the stomach only or involve other structures, i.e., the liver, spleen, kidneys, and intestines. An isolated stomach injury by gunshot or stab is comparatively rare owing to the anatomical position of the organ. When empty, the stomach lies at the back of the abdominal cavity. Its fundus rests under the left cupola of the diaphragm and is covered, in front, by the seventh, eighth and ninth ribs. The body of the stomach is in contact with the left lobe of the liver and only in a small area with the anterior abdominal wall. It can be readily understood that an empty stomach is not likely to be injured as is the liver, colon, small bowel, or the base of the lungs. It is evident that the stomach alone is rarely injured, with other organs remaining intact. However, when the stomach is distended, isolated injuries of this viscus are liable to occur.

The symptoms vary greatly depending upon solitary injury or associated pathologic processes, their extent and character. Whatever the situation, it is certain that each injury to the abdomen demands immediate exploratory laparotomy. The harm done by an unnecessary exploration is outweighed by the risk of a delayed intervention when its indication becomes striking. The time element is greatly important as the mortality rate is considerably increased if intervention is delayed more than six hours. The procedure after the abdomen is opened

depends upon the findings. In an opening in the anterior gastric wall a mere closure in two layers will suffice. If there is an injury to the posterior gastric wall, it will be necessary to divide the gastrocolic ligament, turn the posterior wall anteriorly, close the opening by a two-layer suture, replace the stomach to its normal position, and then suture the severed gastrocolic ligament. If any other structure is injured, such as the liver, spleen, or colon, it is dealt with as described in the chapters concerning the specific organs.

Subcutaneous Rupture of the Stomach

Subcutaneous rupture of the stomach is caused by trauma inflicted upon the body, most frequently the anterior abdominal wall, without causing penetration. That such cases are rare is attested by Sherren's report on 270 cases of subcutaneous rupture of abdominal viscera, as observed in the London Hospital, in which rupture of the stomach took place only five times. Petry, in 1896, collected 219 cases of traumatic rupture of the gastrointestinal tract, of which gastric rupture occurred in thirteen cases. Glassman, in 1929, collected from the literature fifty-two cases of subcutaneous rupture of the stomach, to which he added two cases of his own. Since that time several additional cases have been reported.

The spontaneous rupture may be considered a variety of subcutaneous rupture. The stomach has, seemingly, no pathologic changes in its walls and ruptures without the action of any external force but because of increased intragastric pressure.

INCIDENCE. Subcutaneous abdominal injury occurs more frequently in men than in women (76 per cent) between the ages of four and ninety.

PATHOGENESIS. The force producing the rupture may be direct or indirect. The former may be due to a kick from a horse, squeezing of the abdomen between two cars, or being run over by a car, and the like. Indirect force is sustained by falling from some height on one's feet, buttocks or head, when a full stomach may become ruptured.

PATHOLOGY. Rupture of the gastric wall may be complete or incomplete. The complete kind is most frequent and occurs in about 90 per cent of all cases. In an incomplete rupture the tear may be in the mucosa, or through the serosa and muscularis. As a rule the tears are single, but cases of multiple tears have been reported. An unusual case of complete avulsion of the stomach from the duodenum was reported by Ian Fraser. He treated the patient by blind closure of each open end and reestablished the continuity of the gastrointestinal tract by a posterior gastroenterostomy. The patient made an uneventful recovery. Lifschitz demonstrated that perforation is sometimes extensive. In the case reported by him the entire circumference of the stomach was severed at a distance of 7 cm. from the pyloroduodenal junction. It looked as if it had been

left in the midst of a gastrectomy. The surgeon attempted to unite the severed parts by an end-to-end anastomosis, but did not succeed because the two ends retracted far from each other. He closed both ends and performed posterior gastro-enterostomy. The patient died on the twentieth postoperative day from peritonitis.

Tears occur most frequently on the anterior wall, next in frequency on the posterior wall, and quite infrequently along the lesser curvature.

SYMPTOMS. The symptoms vary in complete and incomplete rupture, and in cases with or without associated pathologic conditions, such as rupture of another viscus or hemorrhage. In incomplete rupture in which the serosa and muscularis are injured, no symptoms are apparent to aid a diagnosis. However, rupture of the mucosa in many cases is followed by bloody vomitus. Hematemesis following a blow on the abdomen or after a fall should make the physician consider injury to the stomach. In fact, many surgeons believe that hematemesis following a blow is pathognomonic of injury to the stomach.

Complete rupture will produce the symptoms described for a perforated gastric or duodenal ulcer, except that there will not be a history of digestive disturbances and the presence of trauma. A flat x-ray plate reveals air between the liver and diaphragm.

DIAGNOSIS. A ruptured stomach should be suspected when there is a history of trauma, symptoms of ruptured viscus, and bloody vomitus.

TREATMENT. The treatment is surgical. While a spontaneous recovery is not impossible it is too rare for therapeutic consideration. The patient should be operated upon, and the sooner the better. The operative mortality is about 80 per cent, and almost all recorded recoveries took place when operation was performed within six hours after injury. The operative treatment in cases not complicated by rupture of another viscus consists of simple closure of the rent. If the rent is in the anterior wall, the procedure is simple; if, however, it is on the posterior wall, the repair may present great technical difficulties especially when the opening is close to the lesser curvature and in the vicinity of the cardia. At this location spontaneous rupture occasionally takes place. If the tear is extensive, it may require more than a simple closure. It is significant that in the few reported cases of complete rupture of the entire circumference of the stomach or duodenum, the severed ends retracted to such a degree that it was impossible to bring them together for an end-to-end anastomosis, so that it was necessary to close each end and to establish the continuity of the gastrointestinal tract by posterior gastro-enterostomy between the proximal portion of the stomach and a high jejunal loop.

Burns of the Stomach

Burns caused by intake of corrosive substances may affect any portion of the digestive tract. They may injure the mouth and pharynx and

do little damage to the esophagus or to the stomach; or they may affect the esophagus chiefly, producing negligible damage to the mouth or stomach. In another group the damage is principally in the stomach, and the other parts of the digestive tract are spared. Of all these possibilities an isolated injury to the stomach is the least frequent, as is illustrated by the fact that up to 1896, Hartman collected from the literature only twenty surgical interventions for isolated burns of the stomach. Quénu and Petit, in 1902, reported thirty-four cases, and Bondareff, up to 1910, added thirty-six. All these were single instances reported by individual surgeons.

In 1933, Tunik reported 8 cases observed in the clinic of Prof. Djanelidze in Leningrad over a period of ten years. In 1935, B. A. Petrov described forty-one cases from the Institute of Urgent Surgery in Moscow from 1929 to 1934. The last group is of particular interest because it covers a great number of cases observed by the same group of surgeons within a reasonably short period; for this reason it is freely quoted here.

Of the forty-one cases as published by Petrov, one-third were confined to the stomach while two-thirds of the patients had burns also of the mouth or esophagus or both. Burns are produced either by the ingestion of acids or alkalis. Intake of acids causes greater damage and, therefore, requires surgery more frequently than that of alkalis in an equal number of cases, the ratio being 3 to 1. But as alkalis are used for suicidal attempts more frequently than acids, the number of patients operated upon for burns by alkalis is about twice that of persons burned by acids.

Another interesting observation is that acids more frequently damage the stomach than the esophagus, whereas alkalis affect the esophagus deeper and more frequently. It has also been observed that inorganic acids (*hydrochloric acid, sulfuric acid, nitrous acid*) produce stricture of the stomach, while organic acids (*oxalic acid, acetic acid*), although very toxic, do not cause damage in the depth of the gastric wall, and thus produce strictures less frequently.

PATHOLOGY. The extent of damage depends upon the following factors: concentration of the corrosive substance, fullness or emptiness of the stomach at the time the poison is taken, posture of the patient before swallowing the poison, first aid measures, and the like. Therefore, all variations from simple swelling of the mucosa to complete gangrenous perforation of the entire thickness of the gastric wall may be observed. Usually the surgeon operates for one of two conditions: First, soon after the accident, when there are symptoms of acute perforation, and second, several weeks to a few months after the accident, when all acute symptoms have subsided and the patient shows signs of stenosis of the stomach.

In the first condition the stomach is small; its walls are dark green and often black, charred and flabby; the serosa has lost its luster and the peritoneal cavity contains sanguineous fluid. In the second condition the picture is entirely different: The antrum is transformed into a narrow tube with a diameter of one-half to one inch; the greatest degree of scar formation is at the point between body and antrum, at the place known as sphincter antri pylorici, or isthmus ventriculi. This point of maximum constriction may be mistaken for the pyloric ring, and the constricted antrum for the first part of the duodenum. However, close examination will reveal the pyloric ring to be distal to this cicatricial tube, facilitating recognition of the tube as the constricted antrum of the stomach. The body of the stomach, constricted at the antrum, has difficulty in evacuating its contents which accumulate and begin to stagnate. Gases are formed which distend this portion of the stomach into the shape of a balloon which may descend down to the pelvis. Therefore, when the surgeon opens the abdomen in such cases, the distended stomach immediately appears in the form of a ball with a hard cylindrical tube separated from it by a constriction. The tube is followed by another constriction which represents the pyloric ring. This picture is constant so that it may be considered as pathognomonic for burns of the stomach producing stenosis.

The corrosive material rarely leaves the stomach and produces burns of the duodenum. However, a few cases have been reported.

SYMPTOMS. The symptoms vary according to the degree of damage done to the stomach and the extent of the associated burns of the mouth, pharynx and esophagus. In cases in which the stomach is not so deeply affected so as to lead to perforation, the patient complains of pain in the epigastric region, which is tender to the touch and vomiting occurs, which is frequently bloody. Mouth and tongue are dry, the patient is toxic and may be unconscious; frequently dysphagia is present. In cases of penetration through the entire thickness of the stomach, all these symptoms are exaggerated and signs of peritonitis appear; the abdomen is tender, the abdominal muscles rigid; Blumberg's sign is often positive (rebound tenderness); the pulse is small and rapid, and the patient is dehydrated and toxic.

TREATMENT. The treatment differs in accordance with the stage of the disease and the damage produced. In the acute stage, without signs of perforation, the treatment is conservative. A large amount of 10 per cent glucose or normal salt solution is administered intravenously; the patient is kept warm, cardiac stimulants are given, if necessary, and also morphine to relieve the pain. If after a few days the patient is able to take fluid and semifluid orally, no further immediate treatment is necessary; otherwise jejunostomy under local analgesia may be performed.

Some patients soon after becoming ill show symptoms of perforation of the stomach. In such cases immediate laparotomy is indicated. The stomach is walled with gauze and brought out. In the few cases on record in which perforation took place, it was impossible to marsupialize the stomach because it became contracted and was lodged high under the diaphragm. It was also impossible to close the rent due to extreme friability of the gastric tissues. Packing of the stomach was not successful because all patients who received this type of treatment died within a few days. It may, therefore, be stated that so far all cases of burns which caused perforation were fatal irrespective of the method of treatment.

An entirely different picture is presented in the treatment of patients in whom the acute symptoms subside and stenosis of the stomach takes place. The patient survives and frequently his health is fully restored. The specific procedure is determined by the following: whether the stomach alone is affected or the esophagus as well is damaged, and the degree of stricture of these organs. If the upper portion of the esophagus alone is involved and regular dilatations are ineffective, gastrostomy should be performed. If only the lower portion of the esophagus is involved and the dilatation is not effective, gastrostomy or jejuno-esophagostomy antethoracica is indicated. If the cardiac end of the stomach is involved, gastrostomy is the proper procedure. If the pylorus is constricted, gastrojejunostomy should be performed. If both ends of the stomach, cardia and pylorus are affected, gastrostomy and gastro-enterostomy are the proper operations.

In cases in which the esophagus and the entire stomach are injured to such an extent that there is little possibility of restoring their lumen or functional activity, jejunostomy is the only possible remedy. Extensive stricture of the stomach has caused some surgeons to perform gastrectomy.

For the technic of these operations the reader is referred to their descriptions elsewhere under the appropriate readings.

The best chance to save life is when the patient is operated upon between thirty and sixty days after sustaining the injury. Prior to that he may still be toxic and after sixty days greatly emaciated.

It should be borne in mind that the different types of technic employed in gastric operations for burns vary from the methods used when the stomach is normal. When the stomach has become cicatricized, it may be contracted and lie high under the left dome of the diaphragm so that it may be impossible to bring it not only outside of the abdominal cavity but even close to the anterior abdominal wall. For this reason it may be advisable to make an abdominal incision parallel to the left costal arch (left Pribram incision), rather than a right or left paramedian incision. In many cases the surgeon is unable to perform a pos-

terior gastro-enterostomy and he has to confine himself to anterior gastro-enterostomy with entero-anastomosis. Sometimes the anterior stomach wall may be too small for the Spivack tubovalvular gastrostomy, and the surgeon should adopt Spivack's method of performing the operation by using also the posterior gastric wall. If conditions render this procedure also impossible, some simpler form of gastrostomy, such as that of Depage, Janeway or Stamm should be carried out. The surgeon should guard against placing the stoma of a gastro-enterostomy close to the injured portion of the stomach, because the latter may cicatrize and involve the stoma which would necessitate a new gastro-enterostomy.

DIVERTICULA OF THE DUODENUM

Diverticula of the duodenum are an object of urgent surgery when they undergo perforation or gangrene or produce volvulus or intestinal obstruction through compression of the gut. To deal effectively with this type of emergency the surgeon has to have a clear conception of the diverticula as to their size, location and relation to the adjacent structures. Although diverticula are not frequently encountered and their complications arise still more rarely, they are found in sufficient numbers to warrant their study.

HISTORY. A diverticulum of the duodenum was first recorded by Chomel in 1710 as an autopsy finding. Morgagni, in 1761, Rhan, in 1796, and Fleischman, in 1815, described duodenal diverticula found in autopsies. Harley, in 1857, Cole and Robert, in 1920, found gallstones in the diverticula. Morrison and Feldman, in 1926, reported a primary carcinoma of a diverticulum of the duodenum. J. T. Case was the first who, in 1913, recognized roentgenologically a duodenal diverticulum.

ETIOLOGY. The incidence varies greatly according to the method of examination. Statistics based on postmortem examination show an incidence of about 5 per cent, whereas roentgenologic observation yields only about 1.7 per cent. The diverticulum may be located in any part of the duodenum, but occurs most frequently in the second portion (66 per cent); next, in the third portion (22 per cent); then, in the first portion (9 per cent) and, finally, in the fourth portion (3 per cent).

A diverticulum may occur at any age but is most frequently observed after the age of fifty.

The diverticula are divided into two groups: (1) **primary**: (congenital, false), and (2) **secondary** (acquired, true).

PRIMARY DIVERTICULA. Primary diverticula are the most frequent type (about 90 per cent). They are formed by a protrusion of the mucosa and submucous layers through a weakened muscular layer of the duodenal wall; for this reason this wall consists only of serosa, submucosa and mucosa. Owing to a lack of a muscular layer the wall is thin and weak so that it is not able to expel the contents of the diverticulum, and its evacuation is made possible by way of the low, depending neck of the diverticulum rather than active peristalsis of its wall.

Primary diverticula are, usually, situated in the second portion of the duodenum and considerably rarer in the third and still more rare in the fourth portion. In the first portion they are so rare that they may be presumed *a priori* to be of the **secondary** type.

Being herniations, primary diverticula originate at the weakest point of the bowel wall which is its mesenteric border. This is the reason why most duodenal diverticula are situated at the concave border of the duodenum, close to the head of the pancreas. They usually extend medially in front or behind the pancreas, in some cases into the pancreas,

and it is then extremely difficult to treat them. Diverticula are, as a rule, situated retroperitoneally, their fundus being the highest and the neck the lowest point. Therefore, drainage is accomplished more by gravitation of their contents than by peristalsis of their wall. The size of the diverticulum varies from a pea to that of a walnut. Occasionally, it may reach the size of a fist and the width of a pencil or merely that of a probe. In the first instance the diverticulum can be readily detected radiologically owing to the easy entrance of barium into the diverticulum.

SECONDARY DIVERTICULA. Secondary diverticula are acquired and usually are caused by traction on the duodenal wall due to some pathologic condition outside the duodenal wall but in close proximity to it (traction diverticula). They may also arise from pressure exerted from the duodenal lumen upon a pathologically changed duodenal wall (pulsion diverticula). Since the pathologic condition causing the diverticula is peptic duodenal ulcer, which usually is situated in the first portion of the duodenum, these secondary diverticula are, therefore, also situated in the first portion of the duodenum. Their wall contains all the layers of a normal duodenal wall.

MULTIPLE DIVERTICULA. Multiple diverticula occur in about 25 per cent of all cases of duodenal diverticula. The additional diverticula may be found in the duodenum or in any other part of the intestinal tract.

Symptoms

There is no symptom or group of symptoms characteristic of a duodenal diverticulum. In many cases the diverticula do not show any symptoms and are found accidentally in x-ray examination, at operation for some other condition, or at autopsy examination.

Despite an apparent diverticulum, the symptoms accompanying it may be caused by some other condition and may, therefore, erroneously be attributed to a diverticulum. Only after careful study excludes pathologic processes of the stomach, biliary system and the pancreas, and fluoroscopic examination reveals that the diverticulum is tender and that stasis is present, one is justified in holding the diverticulum responsible for symptoms.

The symptoms are classified into the following groups:

(1) **DYSPEPTIC GROUP.** The patient complains of fulness in the epigastric region or right hypochondrium, particularly following the intake of food. This is probably due to overdistention of the diverticulum by gastric contents.

(2) **PSEUDO-ULCEROUS TYPE.** This is the predominant type of symptoms. These appear when the diverticulum is inflamed or ulcerated, and resemble those of gastric or duodenal ulcer.

(3) **OBSTRUCTIVE TYPE.** This group manifests retention or even obstructive symptoms of the stomach, produced by compression of the pylorus or duodenum from a filled diverticulum.

(4) **HEPATOBIILIARY TYPE.** The predominant symptoms are colicky pain in the right upper quadrant, selective dyspeptic manifestations and, sometimes, icterus. These symptoms are due to compression of the common bile duct by the distended diverticulum or to an ascending cholangitis.

(5) **PANCREATIC TYPE.** Attacks of sudden pain in the umbilical region which may be accompanied by vomiting and diarrhea are the main symptoms. They are attributed to pancreatitis, secondary to inflammation of the diverticulum, or obstruction of Wirsung's duct by the diverticulum.

Complications

Complications of duodenal diverticula are comparatively rare, and may be classified as follows:

(1) **OBSTRUCTION OF NEIGHBORING STRUCTURES.** (a) **Partial obstruction of the stomach or duodenum.** Clinically, the patient shows signs of partial obstruction of the stomach or duodenum. Upon operation or autopsy one portion of the duodenum appears dilated and the other collapsed. (b) **Obstruction of the common bile duct.** The patient has a dilated common bile duct proximal to the point of obstruction by the diverticulum. There is icterus and other signs of complete or partial obstruction of the common bile duct. (c) **Obstruction of Wirsung's duct.** The few cases which the literature records reveal that the diverticulum compresses the duct close to its fusion with the common bile duct. The Wirsungian duct and its tributaries were markedly dilated. In some cases acute pancreatic necrosis sets in.

(2) **DIVERTICULITIS.** This complication is uncommon, chiefly due to the fact that the neck of the diverticulum occupies the lowermost position and is sufficiently wide for adequate drainage. Nevertheless, if the neck is narrowed, stasis with ensuing inflammation is prone to occur because the wall of the primary diverticulum is very thin and there is hardly any peristaltic movement to expel its duodenal contents. Diverticulitis may, in itself, lead to the following sequels: (a) **perforation of the diverticulum** of which only a few cases are on record. (b) **peridiverticulitis:** in this very rare condition the diverticulum adheres to the pancreas, duodenum, transverse colon, and common bile duct. (c) **duodenitis** and (d) **cholangitis.** The last two conditions occur infrequently.

3. **CALCULI IN THE DIVERTICULUM.** Gallstones are found from time to time in a diverticulum. The first known case, reported by Chomel in 1710, had twenty-two gallstones. Robertson F. Oglivie described a unique case of a diverticulum which contained an enterolith,

Diagnosis

As there are no clinical pathognomonic symptoms, the diagnosis can be suspected only if the patient has signs referable to disease of the upper abdomen, and careful examination of the stomach, gallbladder and pancreas does not reveal any pathologic process.

The diagnosis can be made only by x-ray, although this method has its limitations because the neck of the diverticulum may be so narrow as to preclude the entrance of barium and thus the diverticulum may remain undetected. However, even if a diverticulum has been diagnosed, it does not necessarily imply that this is responsible for the patient's complaints. In about 50 per cent of the cases in which a diverticulum has been removed, the symptoms did not subside. To be reasonably certain that a diverticulum is involved the surgeon has to establish fluoroscopically that there is tenderness and stasis, the latter to be ascertained by an x-ray taken after a barium meal. In all acute pathologic conditions of a duodenal diverticulum which precludes x-ray examination it is impossible to make a preoperative diagnosis of diverticulosis with any degree of certainty.

Treatment

The treatment of a diverticulum is actually that of its complications. It is indicated when the diverticulum causes compression of the adjacent structures, such as the biliary tract, pancreatic duct, or duodenum; when the diverticulum becomes inflamed, gangrenous, or perforated, or when neoplastic changes take place in the structure. There are four different types of surgical treatment, determined by the location of the pathology, i.e., whether it is in the first, second, third, or fourth portion of the duodenum: (1) operation on the diverticulum; (2) sidetracking operations; (3) combined methods; (4) multiple operations.

OPERATION ON THE DIVERTICULUM. The technic varies according to the site of the pathologic process. If the diverticulum is in the first portion of the duodenum, which is frequently the case in secondary diverticula, the latter is resected, alone or together with the first portion of the duodenum or even with the pyloric portion of the stomach. Removal of the first portion of the duodenum is indicated only when there are marked ulcerous changes in that part. In a simple resection of the diverticulum, its neck is clamped by two crushing clamps, cut between the clamps, and the sac is removed. A purse-string suture is then carried out around the stump which is inverted into the lumen of the duodenum. The ends of the suture are tied and a second purse-string suture is placed around the first, thus closing the duodenum in two layers (Fig. 75). In cases in which it is difficult to remove the sac, diverticulopexy may be performed, attaching the fundus of the sac to the proximal duodenal wall.

If the diverticulum is situated in the second portion of the duodenum, which is a frequent occurrence, the diverticulum lies retroperitoneally and close to the ampulla of Vater, and to the common bile duct and main pancreatic duct. The superior pancreaticoduodenal vessels lie in close proximity to the neck of the diverticulum.

The best approach to a diverticulum in the second portion of the duodenum is from the lateral side. The parietal peritoneum is divided later-

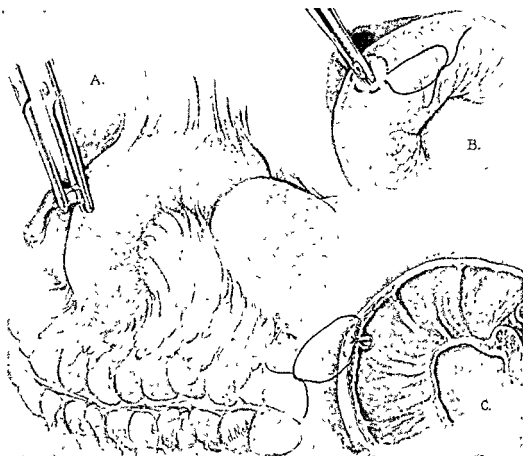


FIG. 75. Duodenal diverticulectomy. A. The diverticulum is cut at its base. B. A purse-string suture is made around the stump. C. The stump of diverticulum is inverted and the purse-string tied. Another purse-string is inserted.

ally and parallel to the second portion of the duodenum; the latter is turned medially so that the posterior duodenal wall faces anteriorly. Thereby, the diverticulum is exposed. It is removed in the same manner as described for a diverticulum situated in the first portion. Care should be taken not to encroach upon the ampulla of Vater.

MacLean described a method which is useful in cases in which the neck is wide and the diverticulum embedded in the pancreas. The anterior wall of the duodenum is opened at its second portion, and the neck of the diverticulum is located. A probe is inserted into the divertic-

ulum through its neck, and the diverticulum is dissected from the pancreatic tissue. When the sac is isolated, it is inverted into the lumen of the duodenum, the neck is ligated, and the sac is cut off. Next, the anterior duodenal wall is closed in two layers. When the diverticulum is in the third or fourth portion of the duodenum, it is approached through the gastrocolic ligament, and resection of the diverticulum is carried out as previously described.

SIDETRACKING OPERATIONS. These are gastrojejunostomy or duodenojejunostomy, leaving the diverticulum intact. The purpose of these operations is to permit the diverticulum to rest by diverting the passage of food which, otherwise, would irritate it. These operations are useful in cases of inflammation of the diverticulum. If, however, there is an impending or actual perforation or gangrene of the diverticulum, or a band extends from it to the neighboring portions of the bowel, side-tracking operations are useless and a direct intervention on the diverticulum is necessary.

COMBINED OPERATIONS. These may be (1) invagination of one diverticulum and resection of another, in cases of multiple diverticula. (2) invagination or resection of the diverticulum, supplemented by gastrojejunostomy; (3) direct attack on the diverticulum, i.e., excision, invagination, diverticulopexy, supplemented by other operations, such as cholecystostomy, cholecystectomy, and the like.

CARCINOMA OF THE DUODENUM

Cases of carcinoma of the duodenum are, usually, divided into two groups: one, which affects the portion of the duodenum above the ampulla of Vater, and the other which affects the part below the ampulla. The clinical manifestations may differ in both groups because the extra-biliary passages (common bile duct, hepatic duct), and the pancreatic ducts (Wirsung's and Santorini's) are earlier and more frequently involved in the first than in the second group. Other symptoms, such as incomplete intestinal obstruction, evidence of metastases, cachexia, are common to both groups.

ETIOLOGY. The disease is extremely rare. It is found in one out of 5,000 autopsies, more frequently in men than in women (ratio 2:1), at an average age of fifty-five but it has been observed in persons twenty-two and eighty-five years of age. Despite considerable literature on the subject, the origin of carcinoma of the duodenum is not definitely known. It has been attributed to duodenal polyps, accessory pancreatic tissue, misplaced remnants of the gastric mucosa and Brunner's gland.

PATHOLOGY. The size of the tumor varies from 0.4 to 8 cm. It may be circumscribed or diffuse. In most cases it is ulcerated. The margin of the ulcer varies from thick and broad to elevated, smooth or honeycombed. The base of the ulcer also varies: in some cases it is smooth, in others nodular; in some the consistency is soft, in others hard. The ulcerated lesions have an annular shape which partially constricts the duodenum, whereas in the non-ulcerated type, the lesion produces almost a total obstruction of the bowel in about half of the cases. Primary carcinoma of the duodenum causes metastases in about 90 per cent. Metastases are found in the mesentery, transverse colon, omentum, liver and lymphatic glands. Carcinoma of the suprapapillary portion involves the common bile duct in 30 per cent and constricts it. This is expressed in symptoms of biliary stasis, such as jaundice, dilatation of the large biliary ducts, proliferation of the small ducts, fibrosis of the portal radicles. In some cases, the neoplasm compresses the ducts of Wirsung and Santorini, producing symptoms of chronic pancreatitis.

SYMPTOMS. In about 50 per cent of all cases, the onset is acute, subacute in about 45, and insidious in about 5 per cent. The symptoms vary according to the extent to which the duodenum is constricted, whether the common bile duct or the pancreatic ducts are involved, and the degree of adhesions to the surrounding tissues. In the acute or subacute onset the main symptoms are epigastric pain which appears in about 50 per cent, vomiting in about 65 per cent, loss of weight in about 50 per cent, jaundice in about 25 per cent in suprapapillary involvement, and in 10 per cent of the infrapapillary type. Anorexia, flatulence, belching, a sense of pressure in the epigastric region are frequent. Later, symptoms of biliary or intestinal obstruction appear.

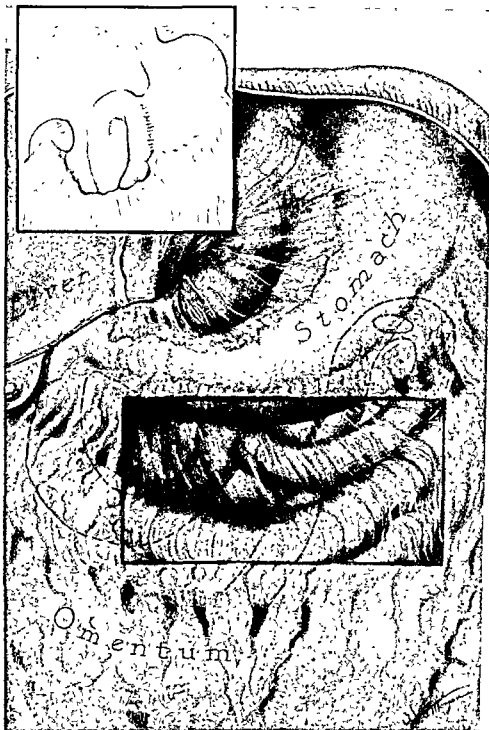


FIG. 76. Palliative surgery of carcinoma of the duodenum. Main picture, Posterior gastrojejunostomy. Insert, Pyloric exclusion.

Palpation of the abdominal wall reveals a mass in one-third of all cases.

Laboratory findings are not pathognomonic. Aspirated gastric contents show blood in 60 per cent and free hydrochloric acid in 50 per cent.

Examination of the feces reveals blood in 65 per cent, acholic stools, in suprapapillary carcinoma, in 40 per cent.

Roentgenologic examination is negative in almost 50 per cent, the reason being that the barium passes rapidly through the duodenum.

DIAGNOSIS. The diagnosis is extremely difficult. Out of about sixty-five cases of carcinoma of the duodenum, a correct preoperative diagnosis was reported twice, and then only roentgenologically. In the rest of the cases an erroneous diagnosis of carcinoma of the stomach, duodenal ulcer, intestinal obstruction, obstruction of the common bile duct and carcinoma of the pancreas were made in cases of carcinoma of the suprapapillary portion. Carcinoma of the small intestine, retroperitoneal tumor and stenosis of the duodenum were diagnosed in cases of carcinoma of the infrapapillary region. Even after opening the abdomen, a correct diagnosis is made only in about 65 per cent.

TREATMENT. The difficulties which confront the surgeon in radical treatment are, primarily, that metastases are present in most cases. These have been found in 75 per cent of cases in which the abdomen had been opened and, therefore, palliative treatment was indicated. In the minority of cases without signs of metastatic involvement, technical difficulties are frequently encountered. There are extensive adhesions from the diseased duodenum into the surrounding tissues, or important structures like the common bile duct, Wirsung's or Santorini's ducts are involved. Under these circumstances radical surgery is feasible only in a few instances. Two operative procedures can be chosen:

Palliative Surgery (Fig. 76). This comprises gastrojejunostomy or pyloric exclusion in which the pylorus is sectioned and the distal end closed blindly, the proximal end being implanted into the jejunum. Cholecystojejunostomy has to be added when the neoplasm involves the extrahepatic biliary tract.

Radical Surgery (Figs. 77 and 78). This is carried out when no metastases are present by removing the diseased portion of the duodenum. If the ampulla of Vater or the lower part of the common bile duct is involved, cholecystogastrostomy should also be made. Owing to its magnitude, this operation should be done in two stages, as described by Allen O. Whipple, William B. Parsons and Clinton R. Mullins.

TECHNIC. The four steps of the first stage are: (1) right upper paramedian or midline epigastric incision; (2) posterior gastrojejunostomy; (3) section of the ductus choledochus, just below the cystic duct and between two ligatures. The surgeon should ascertain whether the cystic

duct is patent so that bile may enter from the hepatic, through the cystic duct, into the gallbladder; if the duct is not patent, the succeeding step (4) should be changed from a cholecystogastrostomy to a hepatogas-



FIG. 77. Radical surgery in carcinoma of duodenum. Common bile duct is cut between two ligatures. Posterior gastrojejunostomy is performed.

trostomy; (4) cholecystogastrostomy: the gallbladder is sutured to the anterior stomach wall, at some distance from the antrum; the opening should be 2 cm. in size.

The second stage of the operation is performed three weeks after the first and consists of the following steps: (1) bilateral transverse trans-

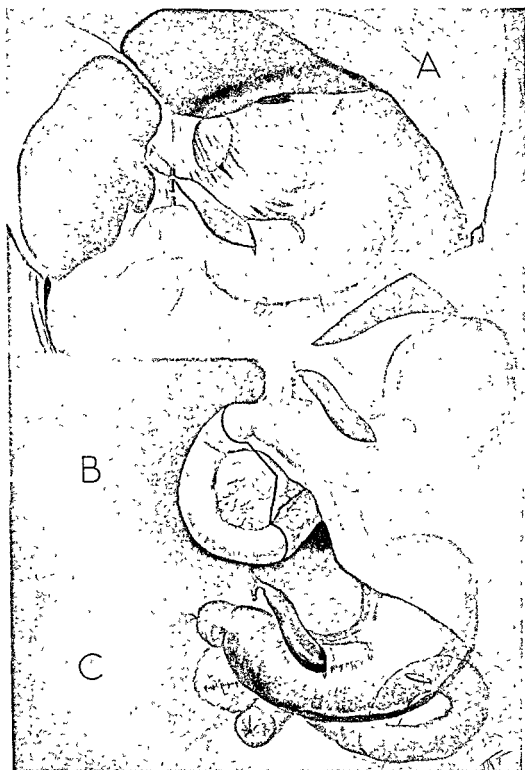


FIG. 78. Radical operation in carcinoma of duodenum: A. Cholecystogastrostomy. B. Excision of the duodenum and of the head of the pancreas. C. Final view.

rectus incision just above the umbilicus; (2) ligation of the gastroduodenal artery; (3) resection of the descending portion of the duodenum, with a V-shaped excision of the head of the pancreas; blind closure of the two remaining ends of the duodenum; (4) ligation of the cut ends of the ducts of Wirsung and Santorini, and suture of the two cut surfaces of the pancreas to each other; (5) drainage of the bed of the resected duodenum with a Penrose drain, and (6) closure of the abdomen.

Non-surgical treatment consists of deep x-ray or, in some cases, radium therapy. The results of x-ray treatment are not satisfactory, and application of radium is objectionable because it requires a laparotomy and duodenostomy to insert the radium.

PROGNOSIS. The prognosis is grave. The immediate mortality is about 40 per cent; 25 per cent succumb within six months, and only 10 per cent live longer than one year.

TRAUMATIC RUPTURE OF THE DUODENUM

Rupture of the duodenum by trauma is a rare occurrence, as compared to that of other abdominal viscera. This is borne out by Guibé's record who was able to collect from the literature only 134 cases observed over a period of seventy-five years (1835 to 1910). These include some cases in which a duodenal ulcer ruptured after trauma, thus reducing the number of cases of a ruptured wall unaffected by a pathologic process. Between the years 1910 to 1940, eighty-four additional cases were reported, a very small number considering the frequency of traumatic rupture of the abdominal viscera. It is considered that rupture of the duodenum constitutes about 6 per cent of all cases of ruptured abdominal viscera due to blunt trauma.

ETIOLOGY. The causes of rupture of the duodenum in the order of frequency are: blows (25 per cent), run-over accidents (22 per cent), kicks (20 per cent), squeezing between hard objects (15 per cent), falls (12 per cent) and other causes (6 per cent).

Any part of the duodenum may be ruptured and the sites of such an accident, in the order of frequency, are: duodenojejunal junction (31 per cent), third portion of the duodenum (21 per cent), second portion (19 per cent), first portion (15 per cent), lower angle (7 per cent), and the remainder (7 per cent).

PATHOGENESIS. There are conditions characteristic for the duodenum which work in opposite directions: one tends to prevent rupture, the other predisposes to it. The former lies in the deep position of the duodenum and its protection by the abdominal wall, with the viscera anteriorly, and by the muscles of the back, spinal column and the ribs posteriorly. These are the natural protectors of the duodenum. However, each portion of the duodenum is separated from the other by angulations: the first from the second by the superior angle; the second from the third by the inferior angle; the third from the fourth by a narrowing; the fourth from the jejunum by another angle. Thus, the flow of the intestinal contents in the duodenum is not as free as in other portions of the intestinal tract and, consequently, the liquid and gaseous contents cannot escape freely to the contiguous portions when struck by some force. The resulting increase in hydraulic pressure is liable to produce rupture. The third and fourth portions are particularly apt to be ruptured because they are close to and in front of the spinal column.

The rupture may occur in the anterior or posterior walls of the duodenum. Rupture of the anterior wall may be intraperitoneal or retroperitoneal, depending upon the affected portion of the bowel. Rupture of the posterior wall is always retroperitoneal. The former occurs twice as frequently as the latter.

SYMPTOMS. The symptoms and their intensity may vary, depending

upon whether there is a rupture of the anterior or of the posterior duodenal wall, the size of the rent produced by the force, whether the bowel was distended or empty at the time of the injury, whether concomitant injuries of other structures were sustained and whether the rupture occurred in the presence or absence of complications. There are no symptoms pathognomonic of duodenal injury and, therefore, the diagnosis is usually made when the abdomen is opened.

In an intraperitoneal injury the patient complains of pain appearing immediately after the injury; it is intense and persistent but not so severe as in gastric or duodenal perforations, and the patient is able to walk unaided, immediately after the injury. The pain is followed by vomiting gastric contents mixed with bile. Tenderness is acute, especially in a small circumscribed area corresponding to that of the rupture. Rigidity is marked in the epigastric region and right hypochondrium, especially in intraperitoneal rupture, but may be mild in retroperitoneal rupture. Usually, the patient is not in shock.

In a retroperitoneal perforation the symptoms are milder: there is no shock; rigidity is not marked; tenderness is moderate. The temperature is slightly elevated, the pulse somewhat accelerated, leukocytosis is moderate; there may be tenderness in the right loin. Nevertheless, the patient gradually becomes sick and toxic, and a spreading cellulitis appears in the right loin. Free air may be observed in the abdominal cavity roentgenologically which, however, merely reveals rupture of an air-containing viscus and is of no help in determining the exact location of the lesion. Hepatic dullness may diminish or even disappear which, likewise, merely signifies the presence of air in the right hypochondrium without revealing its origin.

DIAGNOSIS. The diagnosis of a ruptured gastrointestinal viscus is not difficult; however, it is extremely difficult to diagnose the accident as affecting the duodenum, due to a lack of pathognomonic signs. Yet, rupture of the duodenum may be suspected in a case with a history of a severe blow to the abdomen, especially close to the right and above the umbilicus, followed by symptoms of possible gastrointestinal rupture but without intense pain and the presence of tenderness in the right loin. It is a considerable task to diagnose duodenal rupture before and also after the abdomen has been opened. Many cases have been reported in which the autopsy revealed that a rupture of the duodenum was not recognized at operation, the statistics showing 25 per cent of such cases. There are several reasons for this failure: First, in addition to a ruptured duodenum, there may be a like lesion of the liver, stomach or small intestine, and these may be misinterpreted as the sole injured part in cases in which the surgeon is intent on avoiding prolonged exploration of so deep-seated a viscus as the duodenum. Another reason is that of false security in cases of rupture of the posterior wall of the duodenum,

because in many cases the transverse mesocolon remains intact and the surgeon hesitates to perform such complicated procedures as section of the posterior parietal peritoneum between the transverse mesocolon and the root of the mesentery in order to expose the third portion of the duodenum. To carry out such a manipulation, the following indications are necessary:

(1) The most important is the presence of a tumor mass between the root of the mesentery, transverse mesocolon and the hepatic flexure of the colon. The tumor mass is more or less elastic since it contains escaped gastrointestinal contents, bile, pancreatic juice and gas. It has a shining dark or whitish appearance, depending upon the amount of gas present.

(2) Necrosis frequently occurs in the fat of the omentum, the mesentery, and in that of the retroperitoneal space, and appears as white-yellowish plaques of different sizes. This fat necrosis indicates injury to the pancreas or to the pancreas and duodenum.

(3) In some cases of retroperitoneal rupture gas may escape from the duodenum and spread between the peritoneum and transversalis fascia to the linea alba. It produces emphysema of the anterior abdominal wall and is recognized by crepitus. If the surgeon feels crepitus, while opening the anterior abdominal wall for suspected rupture of a viscus, he should consider a ruptured viscus which likewise has a retroperitoneal surface, i.e., the colon ascendens or descendens or the duodenum. A combination of these signs, namely, tumor mass between the root of the mesentery, transverse mesocolon and hepatic flexure and fat necrosis, together with signs of visceral rupture, renders diagnosis of a ruptured duodenum very probable and exposure of the duodenum imperative.

TREATMENT. The only chance of recovery is in immediate operation. The access is not difficult in an intraperitoneal rupture, be it of the first, second, third or fourth part. If, however, the rupture is retroperitoneal, adequate access to the injured portion should be obtained. If the injury is of the second portion of the duodenum, it can be well exposed by Kocher's method of mobilization (Fig. 79). If it is in the third part, it can be well exposed by a method described by Winiwarter in 1911.

The transverse colon with the omentum are lifted so as to expose the root of the mesocolon. A transverse incision is made through the posterior parietal peritoneum starting at the root of the mesentery just below the center of the root of transverse mesocolon and directed to the right (of the patient) along the base of the transverse mesocolon until the hepatic flexure is reached (Fig. 80, A). The upper lip of the incised parietal peritoneum is reflected slightly upward and the connective tissue in front of the duodenum is exposed. This tissue is bluntly separated until the duodenum is laid bare. The finger then enters between the duodenum

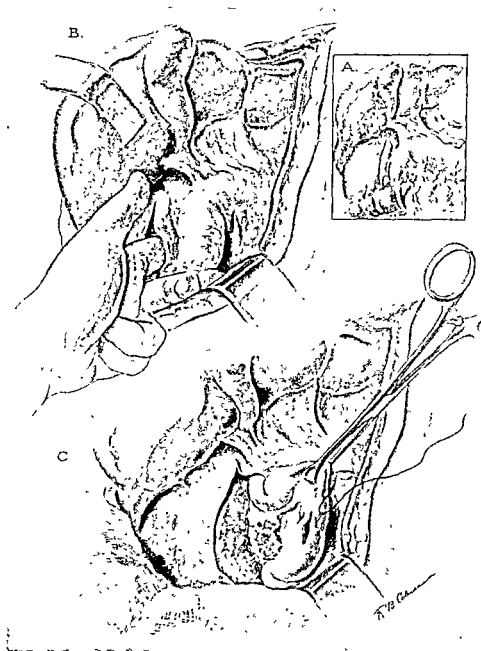


FIG 79 Kocher method of mobilizing second portion of duodenum. A. Location of incision through peritoneum covering the hilum of the right kidney. B. Mobilizing duodenum with index finger of left hand. C. Suturing ruptured posterior wall of second part of duodenum.

and the vertebral column and lifts the duodenum (Fig. 80, B). This mobilization can be made more or less extensive, depending on the size of the injury. If this is small, the opening is closed in two layers and this constitutes the entire operation (Fig. 80, C). If, however, the rent is

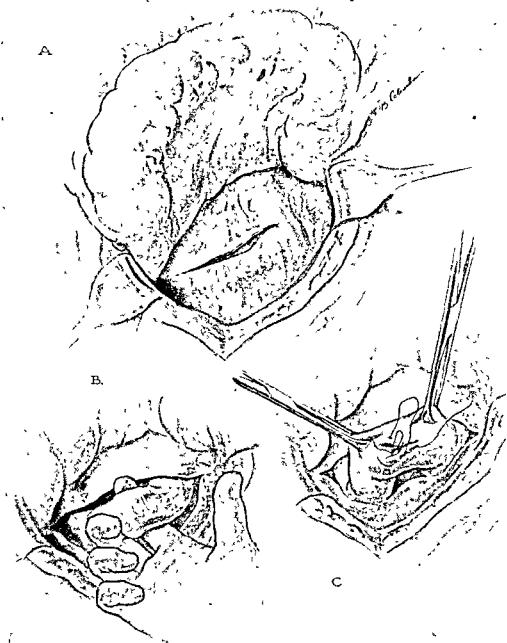


FIG. 80. Winiwarter's technic for repair of injured 3rd portion of duodenum. A Incision through parietal peritoneum of the posterior abdominal wall. B Mobilization of 3rd part of duodenum with index finger after peritoneum has been dissected bluntly from ventral surface of duodenum. C. Repair of rupture in the wall of 3rd part of duodenum by two layers of sutures.

so large that closure of the opening will constrict the duodenal lumen, its closure should be supplemented either by a gastrojejunostomy (Fig. 81, B), if the duodenal stricture is not great, or by a duodenojejunostomy (Fig. 81, A), if the stricture is considerable. If the portion of the duodenal wall is crushed so that simple repair is impossible, it

becomes necessary to resect the crushed portion of the duodenum and supplement this procedure by gastrojejunostomy and duodenojejunostomy (Fig. 81, C).

MORTALITY. The mortality is staggering. According to statistics, compiled by Berry and Giuseppe, it amounts to 87.2 per cent and collected by Hertle—76.8 per cent; whereas, from ninety-four cases recorded by Kanavel, the mortality amounted to 92 per cent. However, between 1910 and 1940, eighty-four cases were reported of which 56 per cent of the

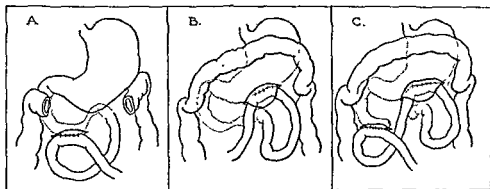


FIG. 81. Winwarter's technic, showing operative steps which may be necessary to do depending on the location and type of duodenal injury. A. Duodenojejunostomy. B. Posterior gastrojejunostomy. C. Resection of the duodenum followed by both duodenojejunostomy and posterior gastrojejunostomy.

patients did not survive. The cause for such a high mortality rate is to be sought for in the late recognition of the injury and the difficulties of the technic.

FOREIGN BODIES IN THE DUODENUM

Foreign bodies in the duodenum become a subject of urgent surgery only when they are so large as to obstruct the lumen of the bowel, or hinder the passage of food through a partially obstructed duodenum, or if the foreign body is sharp and penetrates the duodenal wall. Otherwise, if they are small and have no sharp points, they may show no symptoms and pass *per vias naturales*.

Infants and children frequently swallow foreign bodies, such as beads, marbles, pins and the like; or workers may swallow foreign bodies such as nails and pins which they habitually hold in their mouths. Demented or hysterical adults occasionally swallow foreign bodies. The most frequent are pins, safety pins and nails, but objects such as dentures, the handle of a toothbrush, spoons, and the like are also found. Occasionally, a large gallstone is observed in the duodenum, making its way through a spontaneous cholecystogastrostomy or cholecystoduodenostomy. In isolated cases large pieces of tendinous meat are located in the duodenum as late as fifty days after the meat has been ingested. Bones and fruit stones may pass the pylorus and be arrested in the duodenum. Records reveal a case in which the horizontal part of a T tube was found in the duodenum, inserted into the common bile duct eight years previously. Although there are numerous cases in which a foreign body may lodge and remain in the duodenum, only those which give rise to symptoms draw the attention of the surgeon. This group is relatively small, the number of reported cases not exceeding fifty.

The causes for the arrest of foreign bodies in the duodenum may be classed into two groups: The first is the anatomic characteristics of the duodenum and the possible pathologic changes of its wall; the second group pertains to the nature of the foreign body. Among the anatomic factors causing arrest of foreign bodies is the horseshoe shape with three angles: the *superior*, between the first and second portion of the duodenum; the *inferior*, between the second and third portion, and that at the gastrojejunal junction. Furthermore, the duodenum is comparatively immobile, being retroperitoneal and, in one part, thrown against the spinal column. In marked lordosis, the duodenum may actually form a kink opposite the spine, while, in some cases, the superior mesenteric vessels may partially compress the duodenum. To these anatomic causes may be added pathologic changes in the duodenal wall, such as ulcers and diverticula.

The arrest of a foreign body occurs most frequently in the lower angle and in the duodenojejunal junction.

SYMPTOMS. Foreign bodies in the duodenum infrequently cause symptoms, and are found accidentally by x-ray examination or at autopsy. In the cases manifesting symptoms, these vary according to the character

of the complications, e.g., partial stricture, total occlusion, ulcerations and perforations of the duodenum.

DIAGNOSIS. A foreign body in the duodenum can be diagnosed with a certain degree of certainty only roentgenologically; it is impossible to make a diagnosis clinically. However, even roentgenologically, the diagnosis is not always definitely established.

TREATMENT. When no acute symptoms are apparent medical treatment should be attempted. This consists of the administration of bulky foods, i.e., mashed potatoes or bread, which not only coat the foreign body but protect the mucosa against injury by a sharp or pointed object. When the symptoms point to acute obstruction or impending perforation, surgery should be performed immediately.

The proper operation is duodenotomy and, after removal of the foreign body, closure of the duodenal wall, in two layers, by a series of interrupted or continuous sutures. If the foreign body is lodged in the first or second portion of the duodenum, the operative approach is simple; if, however, the third portion of the duodenum is involved, it should be exposed in the same manner as described for rupture of the duodenum.

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Chapter VII

Congenital Pyloric Stenosis

By JULIUS L. SPIVACK

HISTORICAL NOTE. Fabricius Hildanus (1560-1634) is credited with being the first who in an article under the title "*Observatio singularis de obstructione pylori*," described in 1627 a case in an infant 6 months old with symptoms strikingly resembling pyloric stenosis in infants. As the infant recovered, there is no way of determining whether Hildanus' case was one of true pyloric stenosis.

The second case was reported by Patrick Blair, of Edinburgh, in 1717. It was that of a male infant, who "being a month old was seized with a violent vomiting and a stoppage of urine and stool. Some time after both these became more regular, but the vomiting still continued." The infant died when five months old, and the autopsy showed: "The ventriculus was more like to an intestine than a stomach, its length being five inches and its breadth but one inch. The pylorus and almost all of the duodenum were cartilaginous and something inclined to ossification." Weber, in 1758, recorded a case of a newborn female who "sucked milk without difficulty but soon after she had filled her stomach with this infant nourishment, she returned it by vomiting." The infant died on the sixth day, and the autopsy showed that "The pylorus was hard to touch like cartilage, and contracted. On incision its substance was seen to be thick, and the tightness of its contraction made the lumen similar." Armstrong, of London, in 1771, reported an autopsy made on an infant who was affected with "watery gripes," which revealed, that "There was no morbid appearance to be observed anywhere but in the stomach, and this viscus being so full while the intestines were almost empty, looked as if the disease had been chiefly due to a spasm of the pylorus which prevented the contents of the stomach from passing into the duodenum."

The first case in the United States and the fifth on record was reported by Hezekiah Beardsley, of New Haven, Connecticut, in 1788. On autopsy it was found that "The pylorus was invested with a hard compact substance or scirrhosity which so completely obstructed the passage into the duodenum as to admit with the greatest difficulty the finest fluid; whether this was the original disorder or only a consequence may perhaps be a question."

Siemon-Dawosky in 1842 reported the first case in Germany. Landerer in, 1879, coined the term "congenital pyloric stenosis."

INCIDENCE. Most cases of congenital pyloric stenosis were reported from Anglo-Saxon countries and Germany; considerably less from Latin and Slavic countries. It occurs in negro infants very infrequently. There is considerable preponderance of males over females, approximately at 6 to 1. Most frequently the condition is found among first-born infants, and less often among second, third and even fourth-born infants.

SYMPTOMS. The symptoms usually appear between the third and fourth weeks of age, less frequently between the sixth and tenth weeks; in some cases as late as between the fifth and sixth months.

Vomitus is the most striking symptom. At first it is a mere regurgitation after feeding; later it becomes more forceful and projectile; at the start it is infrequent, but in the course of time appears after each feeding. The child is always hungry, eagerly sucks the finger or the bottle. The feces are scanty. The child becomes emaciated depending in intensity upon the degree of obstruction and the time brought to the hospital. There is decrease of subcutaneous fat and marked dehydration; wrinkling of the skin, sunken appearance of the eyes. Examination of the abdomen shows moderate distention at the epigastric region corresponding to the area of the stomach and flatness of the hypogastric region corresponding to the empty bowels. In the epigastric region one often sees gastric peristalsis. In about 95 per cent a palpable tumor representing the hypertrophied pylorus can be felt. In some cases this tumor can be detected only after repeated examinations and in some cases only under general anesthesia.

DIAGNOSIS. Vomitus, at first in the form of regurgitation and later assuming a projectile type, constipation or scanty stool, loss of weight, visible gastric peristalsis, are very suggestive of this affection. The presence of a palpable tumor clinches the diagnosis as such a tumor is present in no other condition.

DIFFERENTIAL DIAGNOSIS. The following conditions should be differentiated from congenital pyloric stenosis:

1. *Pylorospasm.* The vomitus appears in spells; the interval between the attacks may last several days, when the child appears to be perfectly normal. In hypertrophic pyloric stenosis the symptoms are continuously progressing. There is no palpable tumor in pylorospasm, a condition which is relieved by atropin or its derivatives, such as eumydrin.

2. *Intrinsic types of gastro-intestinal obstruction*, such as atresia of the esophagus, duodenum or small intestine. In this case vomitus appears on the first or second day of life, while in hypertrophic pyloric stenosis it seldom appears before the 9th or the 10th day. If the atresia is below the ampulla of Vater, the vomitus contains bile, in contrast to obstruction of the pylorus or the esophagus when no bile is seen in the vomited matter. In atresia of the esophagus there is often a communication between the esophagus and the trachea above the point of obstruction, which is manifested by respiratory difficulties after intake of food or even the flow of saliva.

3. *Extrinsic forms of congenital obstruction*, as produced by incomplete rotation of the intestinal tract, give signs of obstruction within the first 24 or 48 hours after birth, while in pyloric stenosis, as already mentioned, the signs usually appear only on the 9th day or later. Vomitus also contains bile, which is not the case in pyloric stenosis.

4. *Intracranial injury* or hemorrhage which may occur as a birth trauma may cause frequent vomiting. However, it is not of the projectile

type and there will be signs of brain compression, such as convulsions, spasticity and in some cases bloody cerebrospinal fluid.

TREATMENT. In the majority of cases of pyloric obstruction the condition is attributable to hypertrophy of the muscles and in the minority to pylorospasm. In the latter case the treatment may be medical—feeding with thick cereals and medication. This medical treatment has recently come to the fore again since Svensgaard, of Copenhagen, in 1935, introduced eumydrin (methyl atropine nitrate), 2.5 cc. of a freshly prepared 1.0:10.000 solution being given half an hour before each feed for the first 26 days, after which it is gradually reduced and discontinued after another 15 days. Gastric lavage and subcutaneous introduction of normal salt solution or glucose may be necessary during this treatment. This method yields good results only in cases of pylorospasm, but in obstruction due to true hypertrophy of the muscle only surgery is of value.

Surgical Treatment

The child should not be rushed to the operating table, because pre-operative management to render the little patient safe for surgery is imperative. The infant should be given 100 to 150 cc. of normal saline solution or glucose daily for two or three days during which it is fed by mouth at two-hour intervals. If glucose is given subcutaneously, it should be at three per cent in normal salt solution or as 5 per cent in distilled water. A day before the operation the child should have a blood transfusion, 10 cc. of blood for each pound of its bodily weight. Immediately before operation the infant's stomach is thoroughly lavaged.

THE ANESTHESIA may be general or local. In the former, deep narcosis should never be induced, as very light anesthesia, which will keep the infant quiet, is adequate. While ether, by the drop method, is very efficient, this operation can easily be carried out under local analgesia for which about 15 cc. of a one-half per cent solution of novocain without adrenalin suffices.

OPERATIVE TECHNIC. Many operative procedures have been suggested for congenital pyloric stenosis. They were used with different degrees of success, but all became obsolete after Ramstedt introduced his technic.

1. *Jejunostomy*, was the earliest one performed for the first time for this condition by Cordua, of Hamburg, in 1892. He intended to perform a pylorotomy and the jejunostomy was only to be a preliminary operation for the purpose of feeding the infant to gain strength. However the child died a few hours after the operation.

2. *Gastrojejunostomy*. This was performed for congenital pyloric stenosis for the first time by Carl Stern, of Düsseldorf, in 1897. The child died a few hours after operation. Löbker was the first who successfully performed it in 1898. After that several other attempts were made with the frightful mortality of 60 per cent.

3. *Divulsion of the stomach* (Loreta Operation). James Nicoll, of Glasgow, was the first who performed it in 1899 through a gastrotomy opening. The mortality of this type of operation was about 35 per cent and the results were uncertain.

4. *Pylorectomy*. Only one attempt of this kind was made by Harold Stiles, of London, in 1900. The child died of shock a few hours after operation. No such further attempt became known.

5. *Pyloroplasty* of the Heineke-Mikulicz type was first performed by Braun in 1900. The infant died 20 hours after operation. The first successful case was performed by Clinton Dent, of London, in 1902. Mortality by this method is high—above 40 per cent.

6. *Extramucous pyloroplasty of the Heineke-Mikulicz type*. This was suggested by Fredet in 1907, and Weber in 1910. It consists of cutting the seromuscular layer of the anterior pyloric wall longitudinally and suturing it in a direction perpendicular to the line of the incision. This operation has the disadvantage of presenting technical difficulties since, owing to the marked hypertrophy of the muscle, it is difficult to suture it in a direction perpendicular to the line of incision. However, it served as a stepping stone in the development of an operation destined to become the best method namely, Ramstedt operation.

7. *Technic of the Ramstedt Operation*. The abdomen is opened by a right paramedian longitudinal incision, 2 inches long, starting one inch below the costal arch (Fig. 82, A). The skin, subcutaneous tissue and anterior sheath of the right rectus muscle are cut; the right m. rectus is either retracted laterally or split, the posterior sheath and the peritoneum are opened in the same direction for the same length. Hemostasis should be meticulous and any bleeding point clamped and ligated with very fine catgut. One has to be particularly careful that while opening the peritoneum the small intestine should not protrude outside the abdominal cavity. As soon as the abdomen is opened, the right lobe of the liver is seen. This is lifted with the index finger of the left hand and McFadden's pylorus forceps is introduced to the pyloric portion with the right hand. (Fig. 82, B). Instead of using a forceps one may grasp the pyloric portion with the thumb and the index finger of the right hand. As soon as a hold on the pylorus is secured, the left hand releases the liver and replaces the right hand in holding the pylorus; the index finger and the thumb of the right hand now are exerting a little further traction on the antrum so as to facilitate bringing the pylorus outside the abdomen. A line is chosen on the anterior surface of the pylorus comparatively free of blood vessels; this will usually be midway between the curvatures or closer to the lesser curvature. An incision one-half inch long is now made through the serosa and the superficial muscular layer in the middle portion of the pyloric tumor (Fig. 82, C). From this moment further separation of the muscle is done bluntly, preferably

by the blade of the knife (Fig. 83, D). Though done bluntly, nevertheless the muscular fibers will separate readily until the mucosa is reached, which will pout through the divided muscle and reach the level of the serosa (Fig. 83, E). The points of a mosquito forceps are then introduced

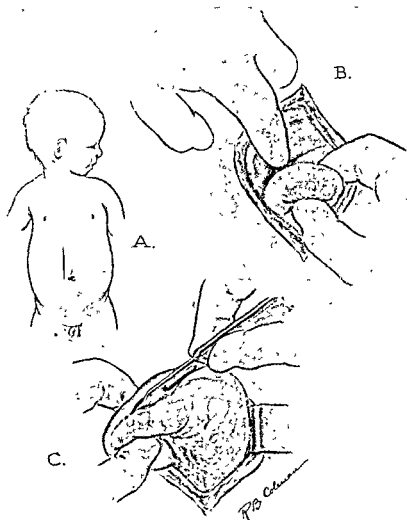


FIG. 82. Congenital pyloric stenosis—Ramstedt operation. A. Abdominal incision. B. Liver retracted, pylorus grasped with right hand and pulled out of abdomen into operative field. C. Pylorus grasped with left hand, incision made through serosa in middle of tumor mass.

between the separated muscular lips and by alternately opening and closing the blades the muscular gap is enlarged at each end close to the duodenum and the antrum. One has to be extremely careful when approaching the duodenal end, as here the mucosa is liable to be punctured, the reason of which will be discussed later. However, one has to be sure that the muscle in the entire length of the pyloric ring extending

from the antrum to the duodenum should be cut so that throughout the lumen is freed of constriction. Usually the procedure is practically bloodless. If there is oozing, it can be controlled by applying warm saline

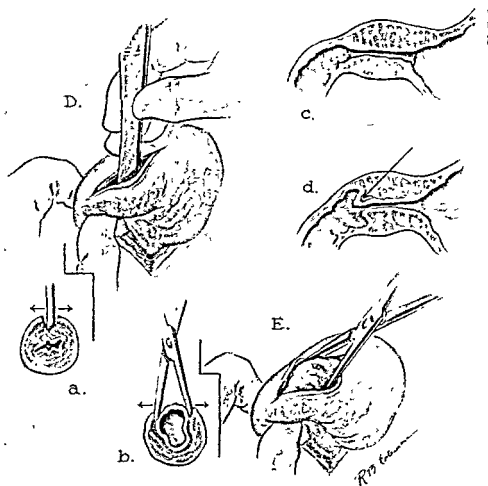


FIG. 83 Congenital pyloric stenosis—Ramstedt operation. D Blunt dissection of muscle fibers with handle of scalpel, a, cross sectional view, E Enlargement of muscular gap in both directions using points of mosquito forceps, b, cross sectional view, c frontal section through usual type of a pyloric tumor; d frontal section through pyloric tumor which has duodenal recess. Arrow (d) indicates place where there is great danger of puncturing mucosa during dissection.

packs for a few minutes. If bleeding persists, the bleeders should be ligated with fine catgut. One now ascertains that there are no perforations of the mucosa and also that the lumen is not constricted anywhere. How to achieve this will be discussed in detail under "complications." After this, the stomach is returned into the abdominal cavity; the ab-

dominal wall is closed in four layers, the peritoneum and posterior sheath being one, the muscle another, the anterior sheath the third, and the skin the fourth. All of these layers should be closed with fine silk in preference to catgut, as the latter necessarily is thick and easily tears the fine layers of the abdominal wall of an infant. A collodion dressing is applied and the abdomen is bandaged.

Complications

There are several complications which may arise after pylorotomy; some of them are attributable to a faulty surgical technic, others are due to general condition of the patient. They may be grouped as follows:

1. Opening of the gastric or duodenal mucosa.
2. Persistence of vomiting.
3. General peritonitis.
4. Hemorrhage from pyloric or gastric walls.
5. Disruption of the abdominal wound.
6. Syndrome of Ombrédanne.
7. Acute liver insufficiency with acidosis.
8. Alkalosis with hypochloremia.
9. Pulmonary complications.

1. **OPENING OF THE MUCOSA** is the most frequent accident in pylorotomy. If it is discovered at the time of operation, no harm results. If the opening is small and remains unnoticed, there will be escape of gastric contents with fatal peritonitis. Therefore, it is imperative, before beginning to close the abdomen, to test whether the mucosa is unbroken. This can be done in the following manner (Fig. 84, A): The first portion of the duodenum is compressed between the thumb, index and middle fingers of the left hand, so as to occlude its lumen; the right hand squeezes the stomach, so that its contents are propelled forward; if the mucosa was not broken, the herniation of the mucosa, already present, will increase; if there is an opening in the mucosa, bubbles of gas and some gastric contents will escape. Two fine catgut sutures suffice to close the opening; for additional safety a small piece of detached omentum may be utilized to reinforce the closed mucosa. Opening of the gastric mucosa may occur even with the most experienced surgeons. The reason for it is that in some cases the duodenal mucosa penetrates deeply into the pyloric tumor mass and if we look through the opened duodenum it resembles the relation of the uterine cervix to the anterior vault as seen from the vagina. It is at this end close to the duodenum where the perforation occurs. The different relations of the duodenal mucosa to the pyloric tumor can be seen in the accompanying illustration (Fig. 83 C and D). To avoid perforating the mucosa one has to be

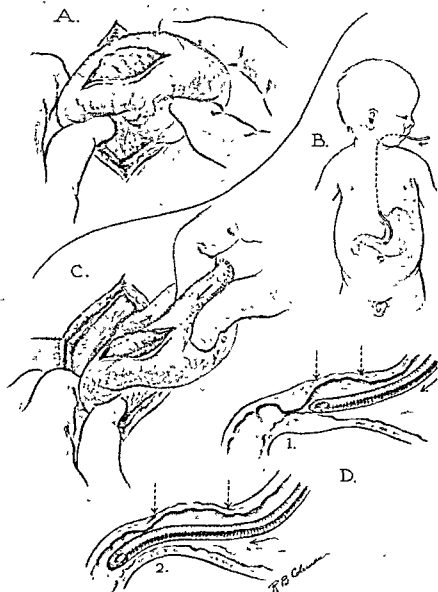


FIG. 84. Congenital pyloric stenosis. A. Method of testing a leak through the mucosa. B, C, D.^{1,2} McLanahan method of testing the patency of pylorus after Ramstedt operation (see text).

extremely careful, when approaching the duodenal end; no sharp division should be made after a knife was used for cutting in the center of the tumor. It is no technical error to leave a few muscular fibers undivided in the deepest layer at the duodenal end, provided that the

patency of the lumen is established. A very helpful maneuver was suggested by McLanahan, namely to push forward through the pyloric canal a catheter, No. 12 French scale, and connect it with a suction apparatus. This catheter he uses before the operation for suction of gastric contents and leaves it in the stomach during the operation. This catheter cannot pass through the pyloric canal when it is hypertrophied but as soon as the constricting ring is relieved, its passage is accomplished without difficulty (Fig. 84 B, C and D).

2. PERSISTENCE OF VOMITUS. This usually occurs if the hypertrophied muscle was not sufficiently cut. However, in some cases it occurs even after complete division of the muscle fibers. Some explain its persistence by hypersensitivity of the gastric mucosa. The administration of sedatives finally overcomes this complication.

3. GENERAL PERITONITIS. This is a very infrequent complication which occurs after inadvertently cutting the mucosa without noticing it. If, however, this accident was noticed and the mucosa closed, no ill results ensue.

4. HEMORRHAGE FROM THE GASTRIC OR ABDOMINAL WALL is extremely rare. To prevent bleeding of the gastric wall, the incision is usually made in a comparatively avascular area and the oozing stopped by hot packing for a few minutes. This usually suffices to control the hemorrhage; if it still persists, the bleeders are ligated with fine catgut; in the few recorded cases of fatal hemorrhage the infants were proved to suffer from hemophilia.

5. DISRUPTION OF THE ABDOMINAL WOUND was recorded several times. It is particularly prone to occur when the abdomen is closed in one layer.

6. OMBRÉDANNE SYNDROME appears as early as at the end of the operation or later within twenty-four hours after the operation. The chief symptoms are:

- a) Elevation of temperature up to 104°F.;
- b) general pallor of the skin and mucous membranes;
- c) tachycardia reaching up to 150 beats per minute;
- d) polypnea in which the number of respiratory movements may reach 50;
- e) profuse sweating; and
- f) extreme restlessness. The child does not sleep and moves incessantly.

This complication as a rule is fatal. Several theories as to its cause were propounded but nothing definite is known so far.

7. HEPATIC INSUFFICIENCY WITH ACIDOSIS is a rare complication. It is manifested by dyspnea of the Kussmaul type, a strong odor of acetone in the respired air, lowered alkaline reserve, lowered concentration of carbon dioxide in the blood and altered concentration of pH in the blood.

The treatment consists of blood transfusion, injection of sodium bicarbonate, glucose, adrenalin and cardiac stimulants.

The prognosis is favorable, if the diagnosis is made early and treatment immediately instituted.

8. **ALKALOSIS WITH HYPOCHLOREMIA** is characterized by paralytic ileus, frequent vomitus, toxic manifestations, dehydration, delirium and fever. The blood shows elevation of the alkaline reserve, hyperglycemia, azotemia and a drop of the chlorides below 2.80 grm. in 1000 cubic centimeters.

If the condition is recognized early, the prognosis is good. The treatment consists of intravenous infusions of 5 to 8 cc. of a hypertonic sodium chloride solution (20%) every 12 hours until clinical improvement and examination of the chlorides in the blood shows that they have reached a normal level.

9. **PULMONARY COMPLICATIONS AFTER THE RAMSTEDT OPERATION** are rare.

Of all the methods employed Ramstedt's is the best. As a matter of fact, hardly any other method now is used. Whereas in the early cases by this method the mortality was about 12 per cent, it is now reduced to 3 per cent, and there are several records of individual surgeons (Donovan, A. Strauss) whose personal experience runs in hundreds of cases, with their mortality less than one per cent.

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Chapter VIII

Gastrostomy

By JULIUS L. SPIVACK

Gastrostomy as an emergency procedure is rarely indicated, but as an urgent operation it should be performed the moment the patient is unable to swallow solid food, that is in all cases where the operation is indicated at all. Delay of carrying out the operation results in the patients' becoming emaciated and therefore poor surgical risks.

HISTORICAL NOTE. Gastrostomy is one of the earliest operations of the stomach. It was first performed by Daniel Schwaben, in 1635, for the removal of an accidentally swallowed knife. It was planned as a gastrotomy, but a gastric fistula was formed, thus creating a gastrostomy. Several cases of traumatic gastrostomy were known in the 18th century; some of these patients lived for decades, taking food through the gastrostomy opening and apparently enjoying good health. To Christian A. Egeberg belongs the credit of being the first to suggest it as a planned operation for feeding patients with an impermeable esophageal stricture. In 1837, he presented a paper on this subject before the Christiania Medical Society. He not only suggested the operation, but gave an outline of its surgical technic, which for many decades was followed by surgeons. Bassow, Professor of Surgery at the University of Moscow, in 1842, repeatedly performed gastrostomy on dogs, and Blondlot at about the same time also did it on dogs for physiologic investigations. The first who performed it on a human being was Sédillot, of Strasbourg, in 1849.

The first successful gastrostomy is usually credited to Sydney Jones of London, whose patient, operated in 1875, lived forty days after the operation.

Several difficulties confronted the surgeons of those days, among which the most important were leakage of the gastric contents and peritonitis.

Among other complications arising from time to time was detachment of the stomach from the abdominal wall and slipping back into the peritoneal cavity. The colon occasionally was mistaken for the stomach and a colostomy instead of gastrostomy resulted. All these difficulties were overcome in a comparatively short time with exception of leakage of the gastric contents. Many ingenious devices were offered against this drawback, and we refer those who wish to familiarize themselves with those methods to the chapter on gastrostomy in Spivack's "Surgical Technic of Abdominal Operations."

Indications

Gastrostomy is performed either for the purpose of feeding the patient when for some reasons he cannot be fed by mouth, or for therapeutic purposes.

A. The usual causes preventing oral feeding are:

1. Constriction of the esophagus due to benign or malignant tumors.

2. Ulceration of the esophagus due to burns by swallowed corrosive material.
3. Strictures of the esophagus produced by healed extensive ulcers.
4. Diverticula of the esophagus, when part of the food passes into a cul de sac, stagnates there and produces esophagitis. In order to control the inflammation, feeding through the mouth should be avoided.
5. Aneurysm of the aorta compressing the esophagus.
6. Tumors of the posterior mediastinum.

B. For therapeutic purposes gastrostomy is performed to facilitate dilatation of the cardiac end of the stomach by a retrograde method.

In most of the cases gastrostomy is done with the aim that the patient will require a stoma as long as he lives. This constitutes **permanent gastrostomy**. In the greatest majority of cases the patients need such a permanent gastrostomy. However, in some cases the patient needs it only for a short time and for these cases a **temporary gastrostomy** is used.

The most commonly used methods are those described by Stamm, Witzel and Spivack.

We use for temporary gastrostomy the method of Stamm or Witzel, and for permanent gastrostomy, Spivack's method.

Witzel's Method (Fig. 85, B)

Described in 1891. A portion of the anterior gastric wall is delivered outside the abdominal cavity. A small opening is made in the anterior gastric wall through which is inserted a rubber tube for a distance of one inch into the lumen of the stomach. The rest of the rubber tube is placed on the anterior gastric wall in an upward direction and to the left; two folds of the gastric wall are sutured over the rubber tube for a distance of $1\frac{1}{2}$ to 2 inches. Thus the opening into the stomach occupies the lowermost portion of the channel and resembles the course of the ureter. Gernez modified Witzel's method in this respect that he placed the opening on the fundus, so that the opening into the stomach occupies the highest point of the channel (Fig. 85, A).

Stamm's Method

Described in 1894. A portion of the anterior gastric wall is delivered outside the abdomen. A circular purse-string suture is made with a radius of one inch (Fig. 86, A). The stomach is opened at the center of this circle and a rubber tube No. 15, American scale, is inserted for a distance of one inch and the tube fastened to the gastric lip by a silk or catgut suture (Fig. 86, B). The purse-string suture is drawn together and the ends are tied (Fig. 86, C). Six interrupted sutures fasten the stomach to the parietal peritoneum; each end of the purse-string suture

is passed through the entire thickness of the corresponding abdominal lip and the ends are tied each to other (Fig. 87, D). The characteristic



FIG. 85. Gastrostomy. A. Gernez modification of Witzel's method (Final view).
B. Witzel's method (Final view).

feature of the Witzel and Stamm methods is that the channel is formed from the anterior gastric wall and lined with serosa, and therefore both have a tendency of spontaneous closure. For this reason they are prac-

tical only when one wants to make a temporary gastrostomy, but unsuitable for permanent gastrostomy in which the opening should remain patent all the time.

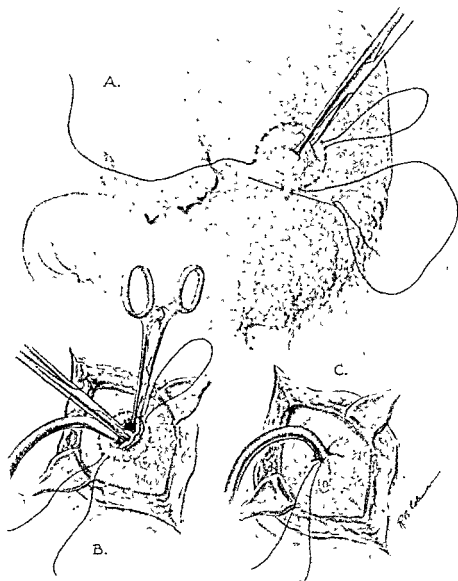


FIG. 86. Stamm method of gastrostomy. A. A purse-string suture is made. B. Insertion of a catheter into the stomach. C. Tying the ends of the purse-string.

Another weak point of these methods is that the gastric contents frequently leak.

In order to overcome these two objections, Spivack, in 1929, devised a new tubovalvular method. This is characterized by forming a tube from a gastric flap of the Depage or Janeway type and a valve at its base. The tube is lined with mucosa and does not become obliterated;

the valve closes the base of the tube, thus rendering the stomach water-tight.

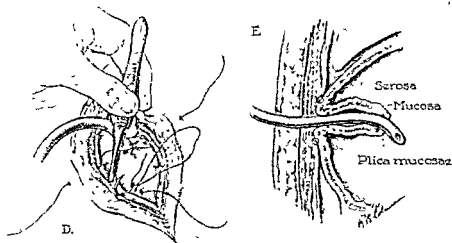


FIG. 87. Stamm method of gastrostomy. D. Fixation of the stomach to the parietal peritoneum. E. Cross section. The channel leading into the stomach is lined with serosa.

Spivack's Method

Step 1. ABDOMINAL INCISION. We prefer to open the abdomen by a left midrectus longitudinal incision, four inches long, starting immediately below the left costal arch.

Step 2. SELECTION OF FLAP. A portion of the anterior gastric wall close to the fundus is delivered outside. A quadrangular flap, three inches long and two inches wide, is outlined and each corner of it is grasped by Allis forceps (Fig. 88, B). If the stomach is small, the size of the flap may be made considerably smaller; however, the ratio of 3:2 (height to width) should be preserved. That the gastrostomy flap may be considerably smaller is attested by the fact that several surgeons, including the originator, made it on infants, children and on the small bowel.

Step 3. FORMATION OF THE VALVE. This is constructed from the upper two-fifths of the flap, thus making the opening close to the lesser curvature; however, in some cases the valve may be made from the lower two-fifths of the flap. The detailed technic of this particular step is as follows:

- a. A seromuscular bite is taken at a point of the line connecting the upper and the lower angles of the outlined flap at a distance of two-fifths of this line from the upper angle. Another seromuscular bite is taken through the upper angle of the same line. Each end of the thread is clamped by a small forceps (Fig. 89, A).

- b. Another suture is placed at symmetrical points along the vertical line connecting the upper and lower angles of the other side of the outlined flap (Fig. 89, A).
- c. A forceps is placed behind these threads and their ends are tied to each other (Fig. 89, A).

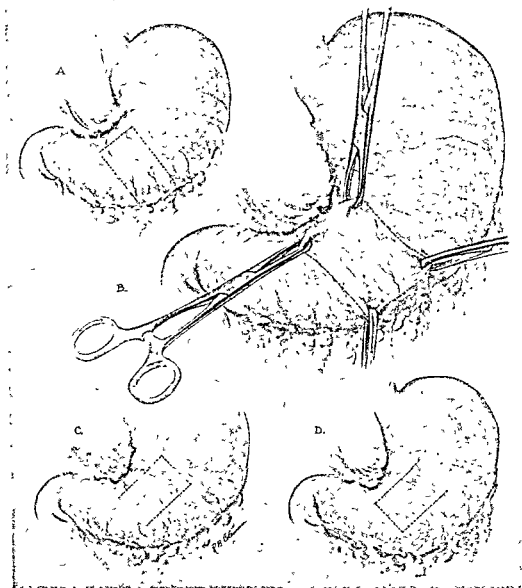


FIG. 88. Spivaek's tubovalvular gastrostomy. A. Depage flap. B. Janeway flap. C. Hirsch flap. D. Reversed Hirsch flap.

- d. A seromuscular suture unites the two opposite ridges, starting from the end, farthest from the operator (Fig. 89, B). Care should be taken in doing so to suture the ridges at symmetrical places and not to encroach upon the lower portion of the flap. (The author saw few cases in which the surgeon doing this step started correctly at one end and while suturing

the opposite ridges reached the lower corner of the opposite side thus leaving no flap for making a tube in the subsequent step.)



FIG. 89. Spivack's tubovalvular gastrostomy. A, B, C. Successive steps in formation of the valve (see text).

Step 4. FORMATION OF THE TUBE.

- a. A seromuscular vertical incision starting from the right upper angle is made up to the right lower angle; another seromuscular incision parallel to the first is made from the left upper to the left lower angle; a transverse seromuscular incision connecting the lower angles is made, Small blood

vessels are seen now traversing the mucosa; they are ligated doubly and cut between the ligatures, thus reducing bleeding to a minimum. Occasionally, we ligate the vessels singly; then by cutting the mucosa, there is some bleeding from the flap, which serves as indication that the blood supply to the flap is sufficient (Fig. 90, A).

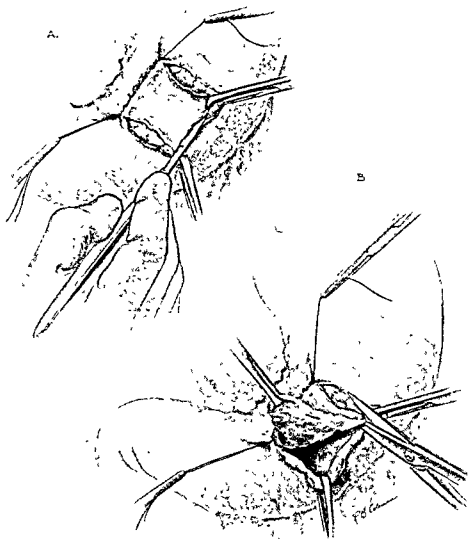


FIG. 90. Spivack's tubovalvular gastrostomy. Formation of a flap. A. Seromuscular incision is made. Blood vessels traversing the mucosa are ligated. B. Cutting the mucosa.

- b. The mucosa is cut along the three lines. The produced flap is reflected upward (Fig. 90, B). At the base of this flap lies the valve, which was constructed in the previous step. Now it is seen that the mucosa of the valve forms a dome, the apex of which reaches nearly to half of the height of the flap—the future tube (Fig. 91, A). When the flap will be transformed into a tube by the subsequent steps, this valve will hermetically shut off the lumen of the stomach from that of the tube.

- c. Suturing the flap into a tube. From a technical point of view this can be done by starting to close the stomach from below; or it can be done by starting the suturing from the upper angles of the flap downwards, until the entire opening is closed. We prefer to do it in the following way:

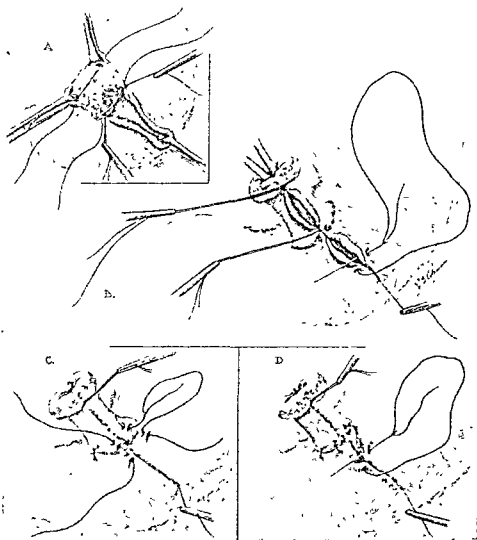


FIG. 91. Spivack's tubovalvular gastrostomy. Formation of a tube from the flap. A. Placing of two interrupted sutures—one at the base and the other at the free border of the flap. B. Previously placed sutures are tied. Closure of the gastric opening. C. Reinforcement of the junction of the base of tube and the stomach. D. Reinforcement of the suture-line by another seromuscular-seromuscular suture.

A bite is taken through each upper corner of the flap; another thread takes a bite through each lower angle of the flap immediately above the point where the seromuscular suture was inserted in step 3 (Fig. 91, A)

The ends of the upper thread are tied to each other; the same is done with the ends of the lower thread. Thus a tube is formed, the upper and lower ends of which are already sutured (Fig. 91, B). The ends of seromuscular suture inserted in step 3 and left long until this moment, are now cut short. Next, the opening into the stomach is closed, starting from the lower end by a continuous through-and-through suture. This suture is continued in an upward direction in order to complete the suturing of the tube, the upper and lower ends of which had been sutured already (Fig. 91, B).

As the valve is very powerful and may even protrude outside the lips of the tube, it is easy to include it in the suture while suturing the lips to each other, which certainly should be avoided because it will occlude the tube. Therefore, it is advisable to place a forceps into the lumen of the tube compressing the valve so as to have it away from the lips of the tube while these lips are sutured, after the completion of which the forceps is removed (Fig. 91, B).

As the point where the base of the tube joins the stomach at the suture line is a potentially weak spot, from where leakage may ensue, it is advisable to reinforce it. This we accomplish by invaginating the base of the tube into the adjoining gastric wall by placing three seromuscular sutures, one bite of which goes through the stomach at a distance of one-fourth of an inch from the base of the tube and the other bite at the wall of the tube at a height of one-eighth of an inch from the base of the tube. This expedient not only reinforces the "weak spot" but may also aid in compressing the wall of the tube against the valve, thus enhancing the watertightness of the stomach (Fig. 91, C).

When the first row of sutures is completed, another seromuscular line of sutures is inserted, also starting from below and reaching the upper end of the tube (Fig. 91, D).

Step 5. FIXATION OF THE STOMACH AND THE TUBE TO THE ANTERIOR ABDOMINAL WALL.

- a. The anterior wall of the stomach is sutured to the parietal peritoneum and the posterior sheath of the rectus muscle by taking a bite through the posterior sheath and the peritoneum of one lip of the abdomen, then through the seromuscular layer of the stomach above the tube and close to its base, next through the peritoneum and the posterior sheath of the rectus muscle of the other lip, when the ends are tied. The same is done to suture the peritoneum and posterior sheath of the rectus muscle of one lip of the abdominal wall, then through the seromuscular layer of the stomach below the tube and then through the other lip, when the ends are tied. Before tying the ends of the thread, care should be taken to see that the tube is not constricted (Fig. 92, A). Thus, two objectives are attained: the stomach is attached to the anterior abdominal wall only at two points and not by a broad surface, and the tube comes to lie extraperitoneally. The latter feature permits the removal of the tube under local analgesia when the temporary stoma is no longer needed.
- b. The wall of the tube is sutured to the anterior sheath in the same manner as described in "a" (Fig. 92, B).
- c. The tube is attached to the skin by four sutures at four points of the tube at a distance of one-fourth of an inch from its free end. One point is at the

anterior wall of the tube, the other at the posterior and the third and the fourth at each lateral side (Fig. 92, C).

The anterior and the posterior points are sutured to both lips exactly the same as the tube is sutured to the anterior sheath, each lateral point

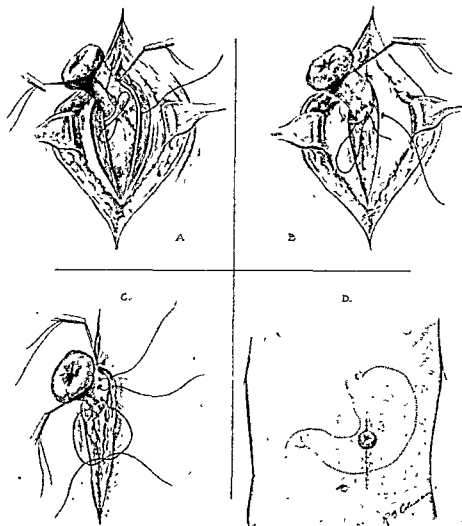


FIG. 92. Spivaek's tubovalvular gastrostomy. A. Fixation of the stomach to the peritoneum. B. Fixation of the tube to the anterior sheath. C. Fixation of the tube to the skin. D. Final view of the opening leading into the stomach.

of the tube being sutured only to its adjoining cutaneous lip (Fig. 92, C). We intentionally leave about one-fourth of an inch of the tube above the skin, to allow for its future retraction. The mucosa of the tube covers the seromuscular layer in the form of a rosette; in this way the serosal layer of the tube does not come in contact with the air, which would cause it to

become dry, the slight secretion from the mucosa of the rosette being just enough to withstand the dryness of the air, but not sufficient to flow upon the skin and irritate it. We do not suture the mucosa to the skin as is

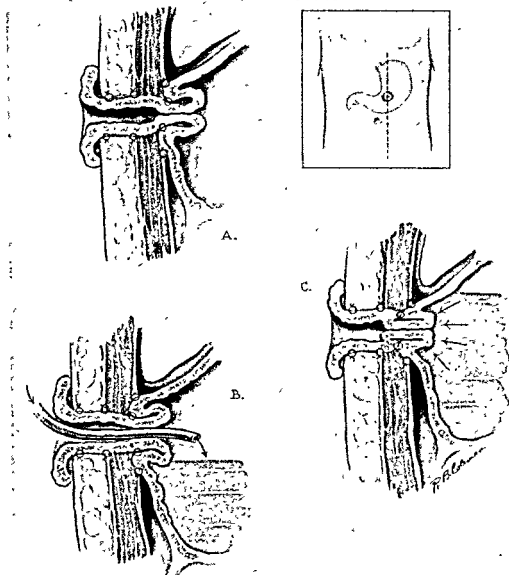


FIG. 93. Spivack's tubovalvular gastrostomy. Cross section. A. The tube is lined with mucosa. The walls of the valve lie close to each other. B. A catheter separates the parts of the valve. C. Increased intragastric pressure brings the walls of the valve closer to each other, thus increasing the watertightness of the stomach.

commonly done by many surgeons the reason for it being that a stitch taken through the mucosa is contaminated and this is one of the most frequent causes of infection of the abdominal wall.

The tubovalvular method can be performed on a very small stomach.

In such cases one does not need to outline a flap of 3×2 inches; it may be considerably smaller. However, the ratio of 3 to 2 (the height to the width) should be preserved. This operation was performed several

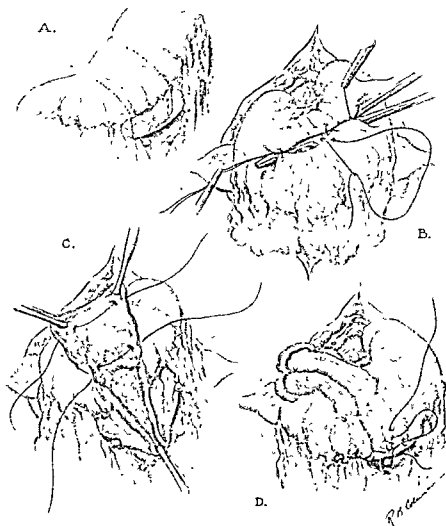


FIG. 91. Spivach's tubovalvular gastrostomy in case of a small stomach. A. Diagrammatic presentation showing that not only the anterior but also the posterior gastric wall are utilized to form a tube and a valve. B. Formation of a valve. C. Formation of a tube. D. Final view (see text).

times on newborn infants and small children and by the originator on the jejunum, colon and urinary bladder.

If the stomach is very small and if one still wishes to have a fairly

good size valve and a tube, one can resort to a modification of this method devised and used by us since 1931, and published in 1933. It consists of utilizing not only the anterior but also the posterior gastric wall, forming the valve from the anterior gastric and the tube from the lower portion of the anterior and from the posterior gastric wall (Fig. 94).

The abdomen is opened and a portion of the stomach is delivered outside the abdominal cavity; the quadrangular flap is outlined as for the usual type of tubovalvular gastrostomy, with this sole difference that the ratio of height to width is 3:4 (in other words, the height is $1\frac{1}{2}$ inches and the width 2 inches); four Allis forceps are placed one at each corner of the flap; the valve is now formed from the entire flap as described in Step 3 of the tubovalvular gastrostomy. The gastrocolic ligament is cut for a width equal to that of the flap; the gastro-epiploic arteries are divided between two ligatures at each corner of the flap; the posterior wall of the stomach is now drawn anteriorly for a distance of one inch and a half, thus forming a quadrangular area, the width of which is two inches and the height—one and one half inches. This posterior wall is cut at its base and sides, thus forming a flap, the edges of which are sutured to each other to form a tube, after which the opening into the stomach produced by cutting the flap is closed. Thus a valve is formed from the posterior gastric wall. The edges of the divided gastrocolic ligament are sutured to each other, thus restoring the normal relation of the stomach to the colon.

We recommend the Stamm method for temporary gastrostomies for a short duration and Spivack's method for temporary gastrostomies of long duration and for permanent gastrostomies.

In a temporary gastrostomy by the Spivack method, the tube may be removed under local analgesia. As it lies extraperitoneally, it will not be necessary to enter the peritoneal cavity.

The mortality depends on several factors, such as whether operation is done for a benign or a malignant lesion, the preoperative management, type of analgesia used and the general condition of the patient. The optimum time of operation is as soon as the patient is unable to swallow solid food. We advise to administer a few days before the operation glucose and normal salt solution intravenously and a blood transfusion a day before the operation, followed by another blood transfusion after completion of the operation.

As a rule we use local analgesia, employing one-half of one per cent of novocain solution with 5 drops of adrenalin (1.0:1000.0) added to each ounce of novocain solution. As a rule, this will suffice. However, if the stomach is small and some pulling is necessary, we supplement the local with splanchnic anesthesia.

With all these precautions the immediate mortality (that is within the following ten days) is about thirty per cent.

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Chapter IX

The Pancreas

By WARREN H. COLE

Lesions of the pancreas requiring immediate surgery are relatively infrequent, largely because it is a solid organ and not subject to danger of rupture, which is so commonly encountered in the hollow viscera. The conditions which may be considered of an emergency nature may be classified roughly into two groups: (1) trauma and (2) infections. Occasionally, infection may be encountered in pancreatic cysts, constituting a condition for which treatment must not be delayed.

Trauma

The location of the pancreas immediately in front of the vertebral column and partially protected by other organs and by the rib margins anteriorly on each side is a major factor in the low incidence of injury. Another factor in the relatively low incidence of urgent lesions lies in the fact that in the relatively minor injuries to the pancreas symptoms may be so mild or so atypical that a correct diagnosis cannot be made. In the former instance when the trauma is mild, consisting only of contusion, failure to operate will not be associated with serious consequences. On the contrary, as will be discussed later, nothing can be gained by operation. In mild injuries, a diagnosis no more accurate than contusion of the abdomen will be possible. Numerous reports of serious lacerations of the pancreas are observed with relatively slight trauma. Likewise there may be no external evidence of injury.

The type of lesion sustained may be classified as follows: (1) contusions, (2) lacerations and (3) perforations. The latter type of injury is inflicted chiefly by gunshot wounds or by stab wounds. In such instances, injury to other organs lying anterior to the pancreas usually will be found. Because of the nature of the pancreatic secretions the pathologic features of traumatic lesions of the pancreas will vary from those noted in injuries to other organs. As encountered in injuries to various organs, lacerations, edema and hemorrhage will be variable. The blood may be present either in the lesser or in the greater peritoneal cavity. Considerable bloody fluid may be found soon after the injury; a large portion of this fluid may represent pancreatic secretions. Two important complications or sequelae to injury to the pancreas are the development of acute pancreatitis and the formation of a pseudocyst. If acute pancreatitis develops, it usually manifests itself within a few days after injury.

If operation is performed later than twenty-four hours following injury, evidence of acute pancreatitis including scattered areas of fat necrosis and induration of the gland may be noted. In addition to **primary hemorrhage** sustained at the time of injury, there is a remarkable tendency toward the development of **secondary hemorrhage** as long as ten days after the injury. This secondary hemorrhage may be massive and is supposedly caused by the digestion of the blood vessel walls by the pancreatic secretions. Just how the trypsinogen of the pancreas becomes activated in the absence of a duodenal perforation is not clear. Numerous instances of **peritonitis** following injury to the pancreas have been recorded; most of these have fatal outcomes. The peritonitis is presumably produced by a decreased resistance of the peritoneum caused by the digestive action of the pancreatic secretions, thereby paving the way for the development of peritonitis, even though the bacterial contamination produced by injury to infected lymphatics, etc., may be trivial. Since the pancreas is buried so deeply, **injury to other organs**, including the colon, stomach, etc., is apt to be present and must be searched for.

CLINICAL MANIFESTATIONS. The manifestations of injury to the pancreas^{1,2,3,4,5,6} differ because of the variation in severity of injury including hemorrhage and because of the frequent development of pancreatitis. Many injuries appear insignificant for several hours but within a day or so manifestations become so pronounced that laparotomy will be found necessary. Even when considerable laceration of the body of the pancreas is sustained there may be relatively little pain and muscle spasm over the upper abdomen during the first few hours following injury. One of the most constant symptoms is **extreme weakness**. This weakness may actually represent collapse or an atypical type of shock in which the pulse rate may not be significantly elevated. It has been suggested that this weakness is caused by injury to the solar plexus. **Nausea and vomiting** may likewise be delayed, but if trauma is significant, these symptoms will almost always be present. Vomiting is occasionally profuse. After a lapse of several hours following injury, **muscle spasm** usually becomes pronounced. Occasionally actual shock develops several hours after injury. In other instances it may develop suddenly several days later. In this instance secondary hemorrhage or rupture of a collection of fluid containing infected pancreatic secretions is probably the etiological factor in the production of shock. Fever is rarely high and develops gradually over a period of a day or two. If peritonitis develops, it will obviously be elevated to 103 or 104° F. and will be accompanied by generalized muscle spasm. The white count is usually moderately elevated. The determination of blood or urine amylase will in most instances be very helpful in making the diagnosis of injury to the pancreas, but only if at least a few hours have

elapsed since the injury. Several hours after injury pain develops and almost always becomes very severe. It commonly radiates posteriorly near the midline. If several days have elapsed since the injury, a mass may be palpable in the epigastrium. This mass may represent a markedly edematous pancreas and adjacent organs, but usually consists of a collection of fluid which ultimately may form a pseudocyst.

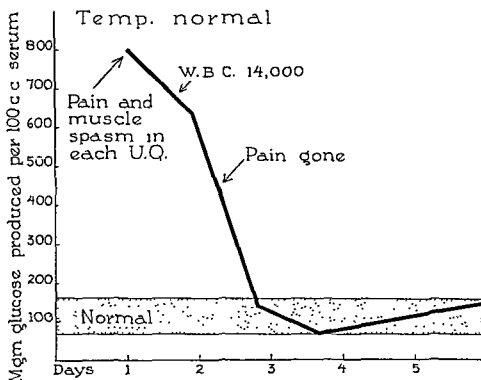


FIG. 95 Blood amylase level in a patient with acute pancreatitis. At entry, 24 hours after onset of symptoms, the blood amylase is elevated far above normal but returns to normal in most instances within 48 hours or shortly thereafter. It must be emphasized then that blood amylase determinations will be of value only if performed within 48 hours of the onset of symptoms.

TREATMENT. The decision as to whether immediate operation is necessary is determined by the appearance of such important manifestations as pain, muscle spasm, and local tenderness, as would be noted in injury to any abdominal organ. In general, the indications are practically the same as for injury of any intra-abdominal viscus. Obviously, if only a contusion is present, operation will not be of any value and may even be harmful. On the other hand, a laceration of the pancreas may result in a fatality because of shock, peritonitis, etc., unless the laceration is repaired. Pain may be so severe as to require morphine as soon as the decision for laparotomy is made. If shock is present, the intravenous ad-

ministration or glucose or blood will be indicated before operation is performed.

A midline incision or a right paramedian incision will be satisfactory for exposure of the pancreas. Palpation should first be done in order to

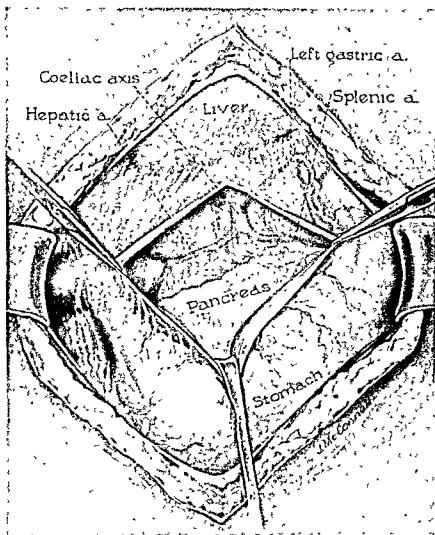


FIG. 96. Gastrohepatic approach to the pancreas. When there is a moderate amount of gastropnoxis and the gastrohepatic omentum long and thin, the pancreas can usually be exposed best by incision through the gastrohepatic ligament as illustrated. The coeliac trunk and branches are shown but are deep enough to be out of the operative field with the exception of the left gastric artery which courses anteriorly to enter the stomach. Care must, therefore, be taken lest this artery or the accompanying vein be injured. However, since ligation of the left gastric artery does not jeopardize the blood supply of the stomach, this vessel may be ligated if by so doing exposure is facilitated.

determine whether or not there is any induration or edema of the pancreas or whether a retroperitoneal hematoma is present. This will give the operator an estimate on the extent of damage. Inspection of the pancreas is best carried out through the gastrohepatic omentum (Fig.

96) or through the gastrocolic omentum (Fig. 97). The choice of approach is determined by palpation and inspection. If the pancreas is found to be located above the edge of the stomach because of a long

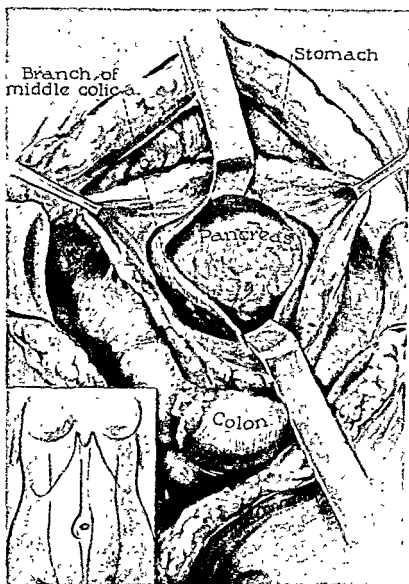


FIG. 97. Gastrocolic approach to the pancreas. When the gastrohepatic ligament is short and fatty the best approach to the pancreas will probably be through the gastrocolic omentum. The pancreas is exposed readily by bluntly dissecting through this structure as illustrated. The middle colic artery will be encountered and must be gently retracted without injury, since ligation of this vessel jeopardizes the blood supply of the transverse colon. The stomach is retracted upward and the colon downward.

gastrohepatic omentum, the best exposure will be through the gastrohepatic omentum. Usually, however, a more direct approach can be made by making a tear in the gastrocolic omentum and enlarging it. A

third avenue of approach through the transverse mesocolon is available but will rarely be feasible because of the edema and hemorrhage in this area. Moreover, there is so much danger of injury to the middle colic artery or one of its important branches, that one of the two approaches

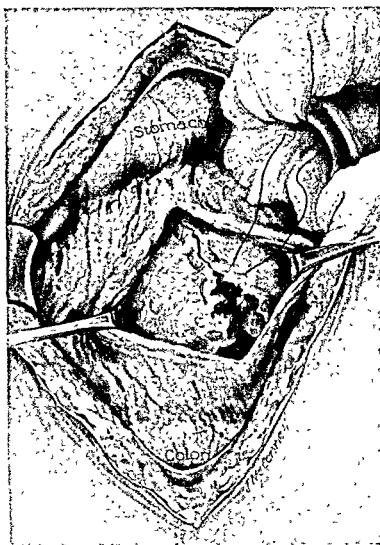


FIG. 98. Suture of the pancreas. When a laceration of the pancreas is sustained, it is extremely essential that the repair be completed carefully so as to eliminate the possibility of a pancreatic fistula. Mattress sutures of the type illustrated will not be as apt to tear out as the ordinary interrupted suture. If the tear extends through the entire organ, posterior sutures must also be taken.

mentioned above is preferable. If exposure of the pancreas reveals laceration, suture of the lacerated area will be necessary. Because of the friability of tissue (Fig. 98), sutures of a mattress type taken fairly deeply are usually preferable. Silk or catgut may be used. If many sutures are required, silk may have a significant disadvantage since prolonged drainage following operation usually takes place.

Naturally, all bleeding points should be controlled. Frequently, it will be impossible actually to grasp these bleeding points with an artery forceps because of the friable tissue unless a larger artery is the source of bleeding. The splenic vein which lies against the posterior border of the pancreas or in the body of the organ may be torn. If this injury has been sustained, a large hematoma will be present. By this time the vein itself will probably be thrombosed; if not, it will still be bleeding and will require ligation. Ligation of the splenic vein should not require splenectomy unless it is done near the hilus, but the spleen should be watched for rapid increase in size immediately following ligation. If there is any question about an adequate flow of blood from the spleen, splenectomy may be indicated because of the danger of spontaneous rupture of the spleen postoperatively. If possible, the pancreas should be rolled anteriorly so as to put sutures also on the posterior surface if the laceration extends through the entire organ. Careful search must be made for injury to other organs. After the local injury has been repaired the wound is closed but a large soft rubber dam or Penrose drain should be placed down to the surface of the pancreas. If a moderate amount of oozing still remains, it may be wise to place oxydized gauze or fibrin foam over the bleeding area. Although the main duct may be torn it will rarely be possible to find the two ends and suture them. Reestablishment of continuity is of course not necessary, but ligation of the distal end is highly desirable to prevent fistula formation.

One of the most important features in the postoperative care is to give the patient no food by mouth for at least two or three days, because food will tend to increase the likelihood of peritonitis and local digestion through stimulation of secretions. An adequate amount of intravenous glucose (3,000 cc. of a 5 or 10 per cent solution) will be necessary. The insertion of a Wungensteen tube with drainage of gastric content to the exterior may lessen the possibility of development of peritonitis. Transfusion should be given if indicated, depending upon the pulse rate and other manifestations of shock.

There is a tendency for a pancreatic fistula to develop following repair of an extensive laceration of the pancreas. However, the digestion of the skin and wound edges is never as severe as it is in the presence of a duodenal fistula since the trypsinogen is not activated. Frequent dressings will minimize the irritation of the skin. Numerous measures have been recommended to control skin irritation. Potter⁷ recommends the application of compresses soaked in tenth normal hydrochloric acid and concentrated beef extract mixed with olive oil. Liquid latex has been recommended, but at least in Smith's⁴ experience was not satisfactory because of the difficulty in preventing secretions from undermining the latex. When the digestion is only slight, the application of zinc oxide ointment along with frequent change of dressings will probably be suf-

ficient to control the irritation although powdered aluminum is much more effective. In severe cases continuous suction of the secretions from the depths of the wound may be the only effective procedure. Fistulae may drain a variable length of time, only a few remaining as permanent. In such instances they may be transplanted into the duodenum or jejunum. Occasionally, the fistula will close but a cyst will form and will require treatment later.

Occasionally glycosuria will develop after the accident. It is quite certain, however, that this complication will occur only in the severe cases or in instances in which the pancreas was largely destroyed after the trauma by scar tissue.

As previously mentioned pseudocysts may develop following injury to the pancreas. It is, of course, possible for actual retention cysts lined with epithelium to develop if the trauma results in obstruction of some of the smaller ducts near the periphery. In a study of forty-eight cases reviewed from the literature, Honigmann⁸ noted that the interval intervening between the accident and appearance of the cyst was extremely variable, but that most of the cysts develop between three weeks and six months following the accident.

Acute Pancreatitis

Because of the lack of agreement as to whether or not acute pancreatitis constitutes a condition for immediate operation, there would naturally be lack of agreement as to whether or not acute pancreatitis should be considered in urgent surgery of the pancreas. However, it is a well known fact that it is extremely difficult to make the correct diagnosis in acute pancreatitis particularly in differentiation from such lesions as a perforated ulcer and a perforated gallbladder. In such circumstances the surgeon will be forced perhaps into performing a laparotomy, and upon finding the condition to be caused by acute pancreatitis must treat this lesion instead.

Although gallbladder disease is an extremely important factor in the development of acute pancreatitis, the latter is much less common even than acute cholecystitis. During a two-year period in the author's experience at Illinois Research Hospital 9.5 per cent of the patients with cholecystitis had also acute pancreatitis, as proved by amylase readings, operative findings, etc. This is, of course, remarkably high and presumably a coincidence; in the two-year period following this interval the incidence dropped to 2 or 3 per cent. The incidence of 3.7 per cent of acute pancreatitis as noted by Heyd⁹ in 557 gallbladder operations would appear to be a more accurate average of the incidence of acute pancreatitis in gallbladder disease.

PATHOGENESIS. A consideration of acute pancreatitis is not accurate unless differentiation is made between the two types, namely, acute

edematous (interstitial) pancreatitis, and acute necrotic or hemorrhagic pancreatitis. As the two names imply, the primary pathologic feature of the former is edema and of the latter, necrosis and hemorrhage. The necessity of discriminating between the two types of pancreatitis has been emphasized particularly by Elman.¹⁰ In either case the gland will be indurated, enlarged and hyperemic. Fat necrosis may be found in either case but is slightly more common in the necrotic or hemorrhagic type. It is very possible that the two conditions merely represent stages in the same process. The theory of reflux of bile as a primary etiological factor was originated by Claude Bernard in 1856. Archibald¹¹ added the theory that spasm of the sphincter of Oddi might aid in the reflux of bile. The production of a communication of the common bile duct with the pancreatic duct as a closed channel produced by the deposit of a stone at the ampulla was demonstrated by Opie.¹² There are numerous investigators, however, who believe that reflux of bile is not the important mechanism in the production of acute pancreatitis. Rich¹³ contends that metaplasia of intraductal cells may produce obstruction of the ducts with subsequent rupture and escape of pancreatic juice into the parenchyma of the gland. Extensive work on the pathogenesis has been reported by Wangensteen¹⁴ and associates, Dragstedt¹⁵ and associates, and others. An important differentiating feature in the pathology of acute edematous and acute hemorrhagic pancreatitis lies in the observation that obstruction of the duct alone (even without reflux of bile) may be capable of giving rise to the acute edematous type. It seems probable that the necrotic or hemorrhagic type is dependent upon activation of trypsin for the degenerative action, which may be so extensive as to involve the walls of blood vessels with consequent hemorrhage. One of the most difficult processes to explain is the activation of trypsinogen in the absence of duodenal secretions. It has been suggested that in some cases bile may be responsible for this activation, or that calcium and other products in the tissue juices may produce it, or even that the pancreas may secrete trypsin in an activated form under certain abnormal conditions.

Attention has already been called to the fact that acute pancreatitis may be secondary to trauma. More commonly, however, it is secondary to other factors, the most important of which is gallbladder disease. Acute pancreatitis is accompanied by gallbladder disease in 80 to 90 per cent of cases. Gallstones are usually present as a part of the cholecystic lesion.

CLINICAL MANIFESTATIONS. The manifestations of the two types of pancreatitis are quite similar except in severity of symptoms. In the acute edematous type, the patient is seized rather suddenly with pain in the epigastrium which radiates to the right or left of the midline and usually posteriorly, particularly toward the vertebral column or to its

left. Nausea and vomiting are variable but usually are not present to a very significant degree. The temperature ordinarily is normal but may be elevated. Examination will reveal tenderness with a moderate amount of muscle spasm in the midline, extending to the right as well as to the left. A moderate leukocytosis is usually present. If the patient is seen early, that is within the first twenty-four or forty-eight hours, diagnosis can be made almost invariably by the amylase test, as popularized by Elman¹⁷ and modified by Somogyi.¹⁸

The manifestations of acute hemorrhagic or necrotic pancreatitis are more fulminating and dramatic than those mentioned above. Commonly the pain comes on suddenly following a full meal. This pain is located in the epigastrium and extends from the right or left and radiates posteriorly. Fever may or may not be present. In a study of twenty-six cases, Fallis and Plain¹⁶ noted a temperature below 100° F. in slightly over half of the cases. This group apparently includes, however, both the edematous and hemorrhagic types. A rise in pulse rate will be present in the more severe cases. Occasionally, the temperature may be subnormal and signs of shock noted. However, Wangenstein,¹⁹ Fallis and Plain, and others have called attention to the fact that even in acute hemorrhagic pancreatitis actual shock is rarely present. In the severe cases mild jaundice will be present in about 25 to 50 per cent. The jaundice is presumably secondary to obstruction of the duct by the indurated pancreas. However, the fact that it is always mild (except in the presence of common duct stones which rarely complicate acute pancreatitis) would point to the possibility that other factors such as hepatitis, hemorrhage, etc., might be responsible for the jaundice. A rise in blood sugar will also be noted in the fulminating cases. This usually is a poor prognostic sign. However, mild jaundice and elevation of blood sugar were recently observed by the writer in a patient who had typical symptoms of moderately severe, acute pancreatitis which was treated conservatively and the patient operated upon two weeks later. At operation an indurated pancreas was found along with gallstones, but there was no evidence that any hemorrhage had occurred. If the case was originally one of acute hemorrhagic pancreatitis, the hemorrhage had absorbed to the point where it could not be identified in any residual form.

As stated previously, at operation an indurated, edematous pancreas will be found in the acute interstitial type; in the acute hemorrhagic type extravasation of blood throughout the pancreas will be noted. There may be considerable blood in the lesser peritoneal cavity and surrounding the pancreas. In the hemorrhagic type there is commonly a small amount of blood-stained fluid in the peritoneal cavity. In over half the instances this fluid will be negative on culture. It was originally thought that absorption of this fluid was responsible for the toxic symp-

toms in acute hemorrhagic pancreatitis. However, as long ago as 1913, Whipple noted that injection of the fluid into the veins and peritoneal cavity of animals was innocuous. These results have been confirmed recently in our clinic by Ireneus.

TREATMENT. It is generally agreed that the treatment of the acute edematous (interstitial) type of pancreatitis should be nonoperative during the acute attack. Since there is no significant destruction of the pancreas, and since recovery takes place fairly rapidly, the institution of conservative treatment is strongly indicated. Since most of the patients with acute edematous pancreatitis have gallbladder disease, it is important that the cholecystitis be treated in order to prevent recurrence of the acute pancreatitis. When the acute attack has subsided, cholecystograms and other examinations for the determination of possible presence of cholecystitis should be performed. If gallbladder disease is found, it is advisable to subject the patient to the same operative therapy that would be indicated in a patient with cholecystitis alone. If acute interstitial pancreatitis is encountered accidentally in an undiagnosed patient who was operated upon for a so-called "surgical abdomen," the judgment of the operator in analyzing the patient's condition will determine the procedure to be followed. In general, if gallbladder disease exists, cholecystectomy or cholecystostomy will be indicated. This problem is discussed in more detail later.

In acute hemorrhagic pancreatitis there is a pronounced difference of opinion as to whether or not immediate operation should be performed. Eminent authorities including Eggers,²⁰ Wolfer²¹ Körte,²² McWhorter,²³ Jones,²⁴ and others are in favor of an immediate operation. On the other hand, equally eminent authorities including Wangensteen,¹⁹ Smead,²⁵ Mikkelsen,²⁶ Hagyard,²⁷ Abell,²⁸ Rapant,²⁹ Demel,³⁰ Nordmann,³¹ and others prefer to treat hemorrhagic pancreatitis conservatively, provided differentiation from perforation of a peptic ulcer or of the gallbladder can be made. By utilizing the nonoperative principle Nordmann was able to reduce his mortality from 50 to 25 per cent in acute pancreatic necrosis. Schmieden,³² who favors conservative medical treatment only when there is no doubt as to diagnosis, has re-emphasized the point, as have Abell and others, that the pancreas must not be incised or torn in an attempt to drain it, as was commonly advised years ago. It is definitely true, however, that during the past few years there has been a trend toward the conservative treatment during the acute attack. The writer agrees with this attitude. However, the surgeon may be driven to emergency operation regardless of his ideas concerning the treatment of acute hemorrhagic pancreatitis, because of the inability to make the correct differential diagnosis. It is quite true that the clinical picture of patients suffering from acute hemorrhagic pancreatitis is very similar to that presented by patients with perforated peptic ulcers, perforated gallbladder, etc.

As an aid in making this correct differential diagnosis numerous observers, including Elman,¹⁷ McCaughan,³³ Mushin,³⁴ Foged,³⁵ Wildegans,³⁶ and others have found the amylase test to be important. Since the time required to perform a blood amylase test is not over two or three hours, it may be performed while operative treatment for the acute condition is being considered. It must be emphasized, however, that the amylase test is of chief value during the first twenty-four or thirty-six hours; in most instances there is a drop in the amylase level to normal after this period. Undoubtedly, many patients with acute hemorrhagic pancreatitis will succumb regardless of what method of treatment is utilized. Wildegans has called attention to the value of the blood sugar determinations in estimating prognosis, and remarks that if the blood sugar is above 300 a fatal outcome will usually ensue.

PREOPERATIVE TREATMENT. While the patient is being observed and an attempt is being made to decide whether or not conservative or immediate operative treatment is advisable, certain therapeutic procedures should be instituted. If more than a few hours have elapsed since onset of the attack and if vomiting has taken place, dehydration will most certainly be present. Only in the very severe cases will actual shock be noted. In either case administration of fluids will be indicated. Although the patient may be complaining of considerable pain, it will usually be unwise to give the patient morphine until the final decision is arrived at regarding conservative or operative care. The application of a hot water bottle or an ice bag to the epigastrium may to some extent relieve the patient. The application of an ice bag is detrimental insofar as at times it increases rigidity or, more accurately, decreases relaxation on the part of the patient.

The administration of 1,000 to 1,500 cc. of 5 or 10 per cent glucose will be indicated. Winslow³⁷ has recently shown 98 per cent of a 5 per cent solution of glucose will be utilized if it is given no faster than 400 to 500 cc. per hour. If the patient has been vomiting considerably, it will be advisable to give Ringer's or Hartmann's solution with the glucose in order to maintain the proper chloride balance. If this is done, 5 per cent glucose should be used because administration of Ringer's or Hartmann's solution with 10 per cent glucose would result in a hypertonic solution. In rare instances, if shock has developed, a transfusion will be indicated. It is important that the patient is not given food of any type because of the stimulation of secretions of the pancreas and other secretions. This order of "nothing by mouth" should be maintained for two or three days or during the time when the manifestations are still acute. It has even been advised that the stomach contents be aspirated to decrease the stimulus toward pancreatic secretions as much as possible, on the basis that the presence of gastric secretions may stimulate pancreatic secretions.

OPERATIVE TREATMENT. Under no circumstances should operation be

performed while the patient is in a state of dehydration or in shock. A high paramedian incision on the right side will usually be found to be most adaptable regardless of the type of therapy contemplated. This would expose the upper part of the abdomen and allow the surgeon to inspect it for such lesions as perforated peptic ulcer, perforation of the gallbladder, etc. If the surgeon is positive that the lesion responsible for the emergency is located in the epigastrium and chiefly in the pancreas, a transverse incision might be more adaptable than a longitudinal one. However, the difficulty in establishing an accurate diagnosis usually makes a paramedian incision preferable.

If the gallbladder, stomach and duodenum are normal and superficial exploration of the rest of the abdominal cavity is likewise negative, the pancreas is then inspected. Accessibility of the pancreas to inspection, etc., is dependent to a great extent upon the amount of fat in the gastrohepatic and gastrocolic omentum, etc. The pancreas should first be palpated to determine if the organ is enlarged or indurated, likewise the area surrounding the pancreas should be inspected for the presence of fat necrosis. Operative approach to the pancreas may be obtained by one of three methods: (1) through the gastrohepatic omentum (Fig. 96), (2) through the gastrocolic omentum (Fig. 97), and (3) through the mesocolon. As stated previously, exposure of the pancreas will usually be attained more readily by making a small opening in the gastrocolic omentum and dissecting downward toward the body of the pancreas. On certain occasions the gastrohepatic omentum will be so long that exposure will most readily be attained by incision through the gastrohepatic omentum. If this method of exposure is chosen, care must be taken to avoid the left gastric artery and vein, which ordinarily are located far enough to the left to be avoided with ease. Palpation of the pancreas will readily inform the surgeon as to which of these two approaches will be preferable. Incision through the mesocolon will rarely be advisable because of danger of injury to the middle colic artery or its important tributaries.

There is a marked difference of opinion as to the type of operative procedure advisable in the presence of acute hemorrhagic pancreatitis. Originally, it was thought advisable to split the surface of the pancreas so that the necrotic contents might drain externally more readily. However, this procedure has been abandoned because the pancreas is divided into lobules by fibrous tissue, thereby preventing drainage of the entire organ; perhaps a more important argument against incising the pancreas lies in the danger of injury to the splenic vein. Four major procedures may be utilized in the surgical treatment of acute pancreatitis:

1. The operative work on the pancreas itself should be confined to simple drainage of the area about the organ, which can be enhanced by

the insertion of soft rubber drain down to this area. The drains should be inserted in such a way that the lesser peritoneal cavity can also drain externally. If this is accomplished, the possible development of an abscess in the lesser peritoneal cavity will be eliminated.

2. In addition to simple drainage of the pancreas, as noted above, **cholecystostomy** is advised by many surgeons in the treatment of acute hemorrhagic pancreatitis. The object of cholecystostomy is to decompress the biliary tract and eliminate any increased tension within the bile tract. One of the disadvantages of cholecystostomy lies in the fact that although this procedure may result in relief from the attack, recurrence is apt to take place.

3. **Cholecystectomy** is advised by some surgeons but should not be performed if the patient is in a critical condition. The advantage of cholecystectomy lies in the fact that the primary common cause of pancreatitis is eliminated.

4. **Choledochostomy** will be indicated if stones are felt in the common duct. Even though no stones are palpable in the duct, choledochostomy and insertion of a T tube in the common duct may be indicated, particularly if jaundice is present. If jaundice is present without stones in the common duct, obstruction of the terminal end of the common duct by the edematous pancreas would appear to be the most likely causative factor. Since the edema subsides relatively slowly, there must be a good theoretical reason for performance of a choledochostomy. As a matter of fact, actual obstruction of the common duct to a degree sufficient in itself (i.e., incident to the edematous pancreas alone) to warrant choledochostomy is relatively infrequent.

In summarizing the treatment of acute pancreatitis, it may be stated that there is practically complete agreement in the conservative treatment of the acute edematous type, and that after the acute attack has subsided, the bile tract disease, if present, should be treated by the usual operative procedures. During the past few years there is a growing tendency to treat acute hemorrhagic pancreatitis in the same way, although some surgeons are still of the opinion that immediate operation is indicated. It must be realized, however, that frequently a correct differential diagnosis between perforated ulcer, perforated gallbladder, etc., cannot be made and immediate operation will, therefore, be necessary. A blood or urine amylase determination will be of great diagnostic value particularly if the patient is seen during the first twenty-four or forty-eight hours of his disease. If operation is performed, any one of the four major procedures described or a combination of them may be resorted to as indicated. One of the most important considerations in the operative treatment is the fact that whatever type of operation is chosen, it should not be so radical as to jeopardize the patient's physical condition. At no time should the parenchyma of the pancreas be split

case there will rarely be difficulty in arriving at the correct diagnosis. The history should reveal the presence of a mass in the upper abdomen of several months' duration, with the development of manifestations of obstruction appearing suddenly or perhaps gradually because of the development first of a partial obstruction. The manifestations consisting primarily of cramping abdominal pain, nausea, vomiting, etc., will vary depending upon the location of the obstruction and its degree. Since they will differ very little, if any, from the usual manifestations of intestinal obstruction, which is presented elsewhere, they will not be discussed in detail here.

The treatment of intestinal obstruction complicating pancreatic cyst may differ considerably from that of ordinary intestinal obstruction. The indications for the administration of fluids and electrolytes and the institution of Wangensteen suction will, of course, be the same. It is particularly important to endeavor to decompress the patient and relieve the obstruction because operation in the presence of obstruction and the cyst would be associated with a higher mortality than with cyst alone. If relief from the obstruction is attained by conservative therapy (i.e., Wangensteen suction, etc.) there is even a stronger indication for laparotomy than in intestinal obstruction associated only with inflammation and adhesions, because of the greater likelihood for additional attacks to develop. Therefore, as soon as the patient has recovered from the obstruction and has been on at least a moderate food intake for a few days, laparotomy should be performed. The incision should perhaps be made directly over the mass, assuming that the intestinal obstruction is related to it. If the obstruction has been produced by adhesive bands, as is usually the case, they should be severed. However, if the patient has recovered from the acute effects of the obstruction, the cyst may be treated in the normal way which in most instances will consist of marsupialization. Although it is conceivable that the pressure of a large cyst on the mesentery of certain loops of intestines may result in torsion with consequent obstruction, the block will usually be secondary to adhesive bands and a thorough search should be made for them.

If the obstruction does not respond to Wangensteen suction, it may be necessary to resort to an immediate laparotomy after a few hours' trial with suction. Rarely will the delay incident to a few hours' suction be damaging, since the process of restoring fluid and electrolytic balance will consume this amount of time anyway.

OBSTRUCTION OF THE COMMON DUCT. Production of obstruction of this type, secondary to pancreatic cyst, would appear to be very improbable. Nevertheless, numerous case reports may be found in the medical literature (Judd and Olson³², D'allaines and Malgras,⁴⁰ and others). Presumably, growth of the cyst extends far enough to the right

actually to compress it; on other occasions tension on adhesions adjacent to the common duct may produce obstruction by kinking. Obviously, the development of jaundice in the presence of a mass in the upper abdomen might have several explanations such as a metastases from a malignant tumor, splenomegalia, absorption from an intraperitoneal hematoma, obstruction of the common duct by stone, etc. Since the patient will rarely be acutely ill when the jaundice first develops, there will be an opportunity to seek diagnostic aid from numerous laboratory measures such as cholecystography, liver function tests, etc. Regardless as to whether or not a definite diagnosis can be made, laparotomy will be indicated unless the jaundice shows evidence of lessening within a few days. Thorough exploration of various organs (including liver, spleen, common duct, as well as the cystic mass) at operation should lead to the correct diagnosis. Treatment will be dependent upon the lesion producing the obstruction.

In Judd and Olson's case the jaundice was apparently produced by pressure of the cyst and was relieved by marsupialization of the cyst. Ten days later a secondary operation, consisting of drainage of infection within the cyst was necessary, but recovery was ultimately satisfactory. The complication of infection in this case was coincidental and would be no more likely to develop than in drainage of an uncomplicated pancreatic cyst.

INFECTED PANCREATIC CYSTS. Because of the proximity of pancreatic cysts to numerous sources of infection such as lymphatics from the intestinal tract, cholecystitis, hepatitis, etc., infection of the contents of a pancreatic cyst might appear to be a relatively frequent complication. Judging from the paucity of reports in the medical literature, however, such a complication is quite rare. In a case reported by Cuny⁴¹ the symptoms incident to the infection developed rapidly. However, in a patient observed by the writer, the manifestations of the infection, consisting of a low grade fever, anorexia, slight weakness, etc., developed insidiously. It is true, however, that when these manifestations of infection were first noted by the patient she simultaneously noted a rapid enlargement of the mass which had been present for several weeks in the upper part of her abdomen. A moderate leukocytosis was present upon admission which was a few weeks after development of the symptoms produced by the infection. This particular patient was treated by marsupialization; recovery was uneventful after several weeks' drainage.

Differential diagnosis in an infected cyst may be extremely difficult. Since infected pancreatic cysts are quite rare, numerous diagnoses must be ruled out. One of the most important of these is an infected hydronephrosis. Frequent urine examination, pyelography and careful examination posteriorly over the region of the kidney will almost certainly rule out this important and likely explanation of the patient's com-

plaints. The development of an abscess about a perforated ulcer must be considered from the differential standpoint. However, since the sequence of events following a perforation of an ulcer is so dramatic, there will rarely be difficulty in ruling out this condition if a careful history is taken. Occasionally, large colloid carcinomas of the stomach become infiltrated with infection and may simulate an infected pancreatic cyst. A correct diagnosis of infected cyst can be made relatively easily if the patient presents the history of a mass in the upper abdomen of several weeks' or months' duration with sudden enlargement simultaneously with the development of the usual systemic signs of infection. Definite though mild tenderness was present in the case observed by the writer and presumably should be present in all cases. The type and virulence of the organism present will naturally determine to a great extent the intensity of the manifestations of infection.

Treatment consists of drainage of the cyst (Fig. 99) as soon as a reasonably accurate diagnosis can be made, particularly as related to exclusion of the possibility of infected hydronephrosis. An incision should be made directly over the mass, splitting the rectus muscle if possible to facilitate drainage. If the mass is not adherent to the anterior parietal peritoneum, a thorough exploration of the mass will be possible. However, since an infection is suspected, all manipulations incident to the exploration should be unusually gentle, lest the infection be spread past the confines of the cyst. An accurate diagnosis will probably not be possible without aspiration. However, before this is done, the area surrounding the site of puncture should be protected by gauze packs and a small caliber needle used. Turbid fluid should not dogmatically be considered infected because the contents of many cysts (rarely in pancreatic cysts, however) are turbid because of the presence of cholesterol. Fluid which is turbid because of the presence of cholesterol will shimmer when held up to the light in a test tube. Microscopical examination will prove or disprove the presence of pus cells. If pus is found, the needle should be carefully withdrawn, the site of puncture painted with iodine and compressed with a piece of gauze to prevent leakage. The peritoneum should be closed at each end and the edges anchored to the wall of the cyst (Fig. 99), so that the peritoneal cavity will not be contaminated when the cyst is opened. The cyst should then be opened and the contents evacuated. After closure of the anterior rectus sheath and skin, the cyst should be packed loosely with iodoform gauze. Because of the presence of infection the opening of the site of the marsupialization should be large, measuring at least 4 or 5 cm. long. Drainage from the cyst should not persist any longer than following marsupialization of an uninfected cyst.

RUPTURE OF CYST. Rupture of pancreatic pseudocysts occurs but is extremely rare. Contrary to expectation, the perforation usually occurs spontaneously and is rarely produced by trauma. Reports of this con-

dition are very scarce in the medical literature. In a study of six cases recently made by Koucky, Beck and Todd,⁴² they noted that the manifestations (severe pain, tenderness, rebound tenderness, rigidity, etc.) were similar to those of perforated peptic ulcer except that true surgical

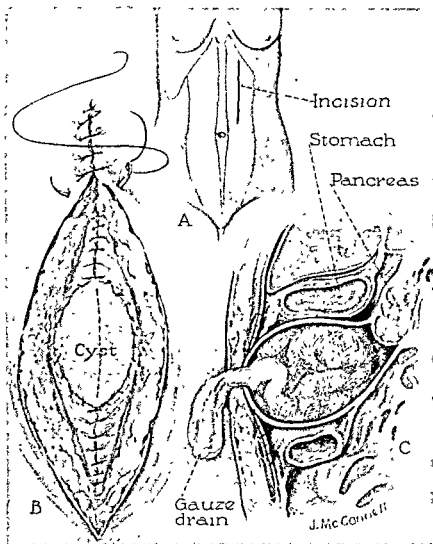


FIG. 99. Management of a pancreatic cyst. When draining an infected pancreatic cyst, it is essential to make the incision directly over the cyst so as to facilitate drainage as much as possible. In most instances a left rectus incision, as illustrated in A, splitting the muscle, will probably be most satisfactory. Before incising the cyst, its wall should be anchored to the peritoneal edge as in B, so as to prevent soiling of the peritoneal cavity. After evacuation of the contents, the cyst is packed with gauze as shown in C, thereby utilizing the principle of marsupialization.

shock was encountered in some of them. A correct preoperative diagnosis will not be possible unless the history of a definite mass in the position of a pancreatic cyst, with its disappearance following development of symptoms, is obtainable. The manifestations should, however, lead to

the diagnosis of a perforated viscus or acute peritonitis due to other causes, thereby furnishing the justifiable indications for operation. In two of the cases studied by Koucky, Beck and Todd, blood was taken at the time of operation for blood amylase determination; in each instance the level was elevated. At operation, the cyst should be marsupialized, preferably through the perforated area. If the perforation is located posteriorly where it cannot be brought up for marsupialization, devitalized tissue at the point of perforation should be excised, the opening closed by suture, and another opening made on the anterior surface for marsupialization. Intravenous glucose and transfusions are important in the preoperative as well as postoperative care.

Miscellaneous Lesions of the Pancreas

There are two lesions of the pancreas which are occasionally associated with hemorrhage: (1) Acute hemorrhagic pancreatitis and (2) carcinoma of the pancreas. The former has already been discussed. Hemorrhage associated with carcinoma of the pancreas is due to deficiency of vitamin K, as recently demonstrated by Dam,⁴² Quick,⁴³ Snell⁴⁴ and others, but rarely develops except as a postoperative complication. Any time during the several days following operation (usually between the third and sixth days) bleeding from the wound may develop. Such hemorrhage may be from the wound edge or from an intra-abdominal source. In either case, the possibility of finding a single large vessel is quite small. A secondary operation consisting of opening the wound is, therefore, rarely indicated. The immediate administration of vitamin K and a transfusion is the treatment of choice. If the patient were jaundiced before operation the prothrombin clotting time should have been determined preoperatively and any delay in it corrected by vitamin K before submitting the patient to operation. It now appears that the administration of vitamin K over a period of four or five days preoperatively will absolutely prevent secondary hemorrhage if it is caused by vitamin K deficiency. If vitamin K has not been given preoperatively and hemorrhage develops, the vitamin (in soluble form) may be given intravenously or subcutaneously, preferably the former. By this procedure a delayed prothrombin clotting time can be corrected within an hour or two; hemorrhage should also cease within that time unless the bleeding is due to another cause such as incomplete hemostasis.

On rare occasions intestinal obstruction may be secondary to lesions about the pancreas. Obstruction associated with pancreatic cyst has already been discussed. As stated previously, the formation of adhesions subsequent to acute pancreatitis may be sufficiently pronounced as to produce obstruction, but only on rare occasions indeed. In the terminal stage of carcinoma of the pancreas, when metastases have disseminated throughout the peritoneal cavity, intestinal obstruction may be pro-

duced by the infiltrating metastatic areas. Such a complication occurs so late, however, that the patient is apt to be in a condition considered hopeless. Commonly, exploratory laparotomy has already been performed and the hopelessness of the situation known. If the patient is not in a precarious condition, laparotomy to correct the intestinal obstruction may be necessary. Conforming to the principles outlined elsewhere, preliminary treatment with Wangenstein suction may be advisable.

CARCINOMA OF THE HEAD of the pancreas results in obstruction of the common duct with consequent dilatation of the duct. It would appear then that rupture of the common duct with subsequent development of mild peritonitis might take place. Rupture of the duct through the wall not invaded with carcinoma rarely takes place, although the writer recently observed a patient with an abscess located just to the right of the duodenum, and presumably due to a perforation of the common bile duct secondary to a carcinoma of the ampulla of Vater; the tumor was resected at a later date after the infection had cleared. On extremely rare occasions when the carcinoma has spread up along the common duct, perforation may take place through an area invaded by the tumor. If such a perforation should develop, signs of peritonitis varying markedly in degree will manifest themselves. Manifestations including pain in the upper abdomen, nausea and vomiting, tenderness, muscle spasm, etc., will develop and may lead one to a decision to operate. If such a condition is found at operation, very little can be done to remedy the situation. If the gallbladder is not very badly diseased and the cystic duct appears potent as well as proximal to the tumor, the common duct may be ligated above and below the perforation, if possible, and anastomosis made between the gallbladder and pylorus or duodenum. If the gallbladder cannot be used for establishing an anastomosis, the other alternative would be to anastomose the free end of the common hepatic duct to the pylorus or duodenum. If the patient is in a critical condition, nothing may be indicated except establishment of drainage of the bile to the exterior.

During recent years ADENOMA OF THE PANCREAS with production of hypoglycemia has attracted considerable attention, chiefly because the symptoms of hypoglycemia are so dramatic. Obviously, when the symptoms have progressed to the point at which coma and convulsions are present, something urgent must be done. However, since administration of intravenous glucose is so effective in restoring the patient's consciousness and normal mental balance, this is naturally the procedure of choice. Operation should be postponed until the hypoglycemia is under control and until the patient's physical condition justifies laparotomy for search of the adenoma.

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because it contains the common duct, the hepatic artery with its branches, the cystic artery and the portal vein.

SIZE AND FORM. The liver is the largest viscus of the human body with an average weight between 1,500 and 1,700 grams, this means one-

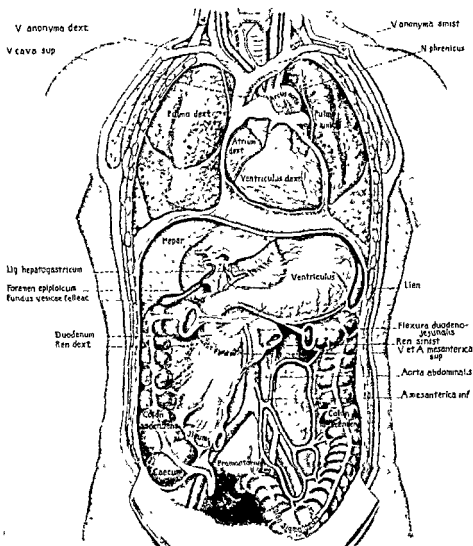


FIG. 100. Position of the liver in relation to the neighboring organs

fortieth to one-thirty fifth of the adult body weight. The size of the liver varies considerably under different conditions. The reason for this variation in size is due not only to the stasis of blood and venous con-

gestion in the case of a failure in the right portion of heart and not only to the stasis of bile in the case of an obstruction of the common duct, but even more frequently to an acute fleeting edema.

This edema must sometimes be considered as a collateral edema in the case of an acute inflammation of the gallbladder, in other cases as a form of allergic edema during digestion when there exists an intolerance to food and in still other cases as the first symptom of an acute hepatitis.

The size of the liver diminishes sometimes under our hands during

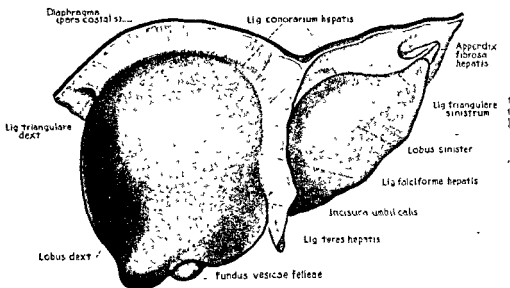


FIG. 101. Ligaments of the liver.
(After Toldt)

the operation. This most important fact, which we shall mention later, must also be considered when discussing the surgical anatomy.

While these three factors determine a transitory change in the size of the liver, an increasing or shrinking of the fibrous tissue causes a rather permanent change in its size.

Changes in the form of the liver are also due to so-called plasticity, so that the surrounding organs may have a moulding influence.

Flattening or relaxation of the diaphragm can transform the convexity. This can be proved when injecting air into the abdominal cavity and x-raying the patient. It can be seen that the air collects between the diaphragm and liver and that the superior convex surface can be flattened to such a degree that the organ becomes triangular in shape.

A tumor of the gallbladder may change the formerly shallow form of the liver bed. The right upper pole of the kidney may cause an impres-

sion. Also the so-called hepatoptosis is not really a ptosis but rather a transformed and elongated form. The moulding influence during the fashion of lacing was well known in former times.

Topography of the Biliary System (Fig. 102)

We distinguish between the intrahepatic and extrahepatic bile ducts. The intrahepatic bile ducts begin as so-called bile capillaries, which are

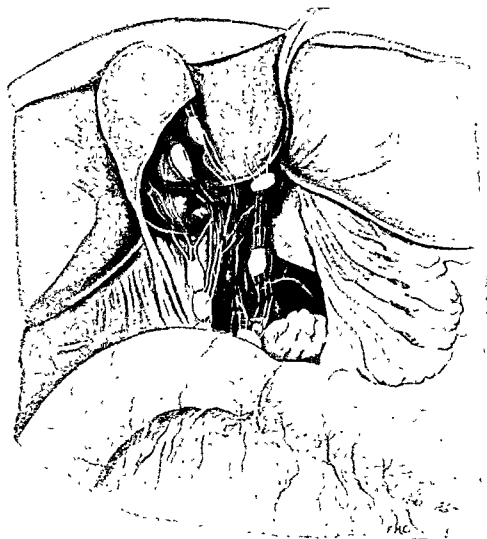


FIG. 102. Blood vessels and lymphatics of the gallbladder and liver.

mere spaces between the liver cells without any special wall. This fact is important because we cannot speak of a real cholangitis, meaning that the smallest bile ducts are infected.

An infectious process in the bile capillaries must always be considered as involving the liver cells, as a parenchymatous hepatitis. The junction, where these intercellular bile spaces become real capillaries with a wall, is a favorite place for inflammatory reaction.

The bile from these small ducts empties into the interlobular ducts and finally into two main right and left intrahepatic ducts. They unite into the extrahepatic common duct, generally called the hepatic duct, bearing this name until it joins with the cystic duct. From this junction downward the common duct is usually called ductus choledochus or the common bile duct. The gallbladder connected by the cystic duct with the common bile duct must be considered as an overflow reservoir which fills when the sphincter muscle on the papilla is temporarily closed. This is especially the case during fasting. The gallbladder empties through the cystic duct when, after a big meal, the sphincter muscle relaxes.

The position of the gallbladder can be defined on the surface as the place where the lateral border of the right rectus muscle reaches the costal arch. (This place is approximately the junction of the tenth and ninth cartilage.) It is in the average person situated two to three inches to the right from the median line. The fundus of the gallbladder is, when protruding from the lower border of the liver (which is the case in 55 per cent according to Gilbert and Partourier), in contact with the peritoneum of the abdominal wall.

We distinguish in the gallbladder fundus, the body and the neck. The body of the bladder is directed anteriorly, downward, to the right. The direction of the neck is to the left, upward to the porta hepatis.

The normal length of the gallbladder is about three to four inches; the average content about 50 cc.; but the size of the gallbladder varies considerably through pathological processes. The body of the bladder is in the majority of cases fixed and imbedded in the liver bed. The part protruding beyond the lower surface of the liver bed is covered with peritoneum, whereas the part closely connected with the very liver parenchyma in the liver bed is covered with fibrous tissue only. When removing the bladder, a wounded region of varying size remains in the liver where the liver tissue lacks any covering.

The fixation of the gallbladder to the liver bed varies considerably. We find all forms from an entirely intrahepatically situated gallbladder to a freely movable gallbladder connected only with a small band which is even pedunculated only on the cystic duct and the cystic artery. This latter but rare type is of pathological importance as it predisposes to axis torsion.

The following diagram gives an illustration of this various relationship of gallbladder to liver (Fig. 103).

An abnormally movable gallbladder may, due to the gravitation, sink

into the lower parts of the abdomen forming a kink with the neck or the cystic duct. We speak of a ptotic gallbladder which sometimes has difficulty in emptying its content.

The neck of the bladder is usually entirely intraperitoneal and comes frequently into close contact with the common duct. Sometimes the neck is situated entirely behind the common duct, that is to say, that the common duct crosses this part of the bladder, while the cystic duct

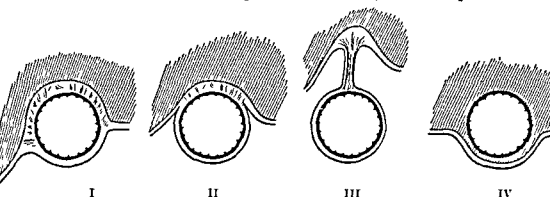


FIG. 103. Position of the gallbladder in relation to the liver. I. Firmly fixed bladder II. Average bladder. III. Loose and movable bladder IV. Intrahepatic bladder

empties into the common duct from behind. These abnormalities are of importance for the surgeon and will be illustrated in the following diagrams.

The Wall of the Gallbladder

The wall of the gallbladder is, under normal conditions, thin but firm. Increased pressure in the gallbladder, due to an obstruction in the cystic duct and stasis, results in a dilatation of the wall without the wall being weakened.

Friability of the wall is always a sign of pathological conditions due to an inflammatory process. In chronic inflammation fibrous tissue increases and the wall may be thickened to $\frac{1}{2}$ cm. and even to 1 cm.

In old cases in which the inflammatory process has come to a standstill, a definite tendency to shrink is observed. The gallbladder can then be reduced to the size of a walnut or even a hazelnut.

The normal color of the gallbladder is bluish-white when emptied, but when filled with bile the appearance is more or less green.

Anatomically we distinguish, as in the intestines, three layers in the wall of the gallbladder: mucosa, muscularis and serosa.

On the mucosa we observe some folds even in the distended state, slippery to the touch, due to the secretion of mucus from the epithelium. Microscopically we see a lamina propria lined with a high, cylindrical, secreting epithelium. The lamina propria contains also fibers.

The muscularis consists of layers of plain muscles intermixed with

fibrous tissue. We know from physiological experiments that electric irritation of the gallbladder does not result in a marked contraction.

The emptying of the bladder is not the consequence of a real peristalsis but rather of a general increase of the tonus of the wall. But the form changes during the process of emptying, becoming more slim and elongated.

The *serosa* (peritoneum) is connected by loose fibrous layers with the muscularis. Under normal conditions the serosa can easily be separated from the muscular layer (subserous cholecystectomy). We can remove the whole bladder within the layer between muscularis and submucosa quite easily and without injury, provided the inflammatory process has not resulted in a fusion of all the layers. The fibrous tissue surrounding the neck of the bladder fixating this part to the liver is usually firm. This firm fixation of the neck of the gallbladder to the liver is due partly to the fact that small branches of the cystic artery may pierce directly into the liver parenchyma. Also bile ducts of different caliber can empty directly from the liver into the gallbladder. This fact is important and is sometimes the cause of a postoperative leakage of bile after cholecystectomy.

Congenital Abnormalities of the Gallbladder

A few words regarding abnormalities may be in order since there is practically no organ which is so liable to variations than the gallbladder, a fact which may be of surgical importance in any case. The following are the main abnormalities observed:

(1) Absence of the Gallbladder:

- (a) Gallbladder may be entirely absent
- (b) Gallbladder may be rudimentary
- (c) Gallbladder is represented by a fibrous cord

(2) Double Gallbladder:

- (a) Two completely separated gallbladders
- (b) A bilobed gallbladder with a septum dividing it into two parts

(3) The gallbladder can be separated by transverse septa into different chambers, in most cases into three chambers. These chambers can be connected sometimes only by narrow tracts. This abnormality is rather frequent and is important because the emptying of such a bladder is often incomplete. Stasis and formation of stones is the sequel. A rare case is the total absence of the cystic duct in which a singular hepatic duct empties into the bladder so that the removal of the bladder with its only outlet results in dividing the main duct.

Such cases will be discussed in the chapter on injuries to the bile ducts.

CYSTIC DUCT. The cystic duct is buried with the end of the neck of

the bladder in the hepatoduodenal ligament, and is usually surrounded by a considerable amount of fat.

When dividing the serous and fatty tissue at this part, the firm tissue of the cystic duct can easily be laid bare. It forms a reverse figure of S,

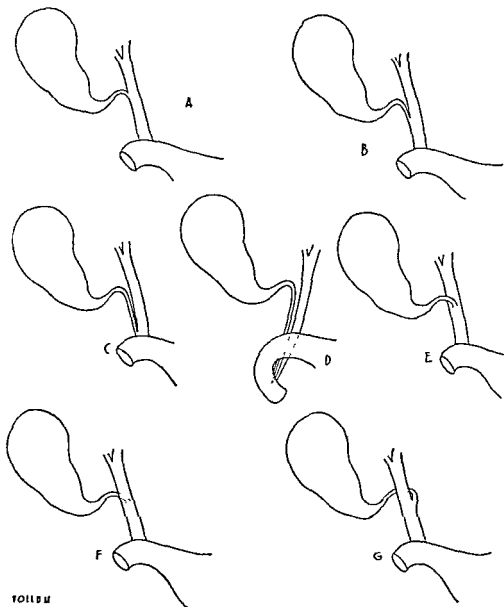


FIGURE 104

FIG. 104. Variations in the course of the cystic duct. (From Julius L. Spivack's "The Surgical Technic of Abdominal Operations," 4th edition, 1946. Courtesy of Charles C Thomas, Publisher, Springfield, Illinois.)

but many variations in the normal kinks and bending of the cystic duct are observed. The course of the cystic duct makes it impossible or at least extremely difficult to introduce even the finest probe from the neck of the bladder through the cystic duct into the common duct (Fig. 102).

It is obvious that this complicated course of the cystic duct with its loops and kinks and internal valves may easily give rise to difficulties in emptying the bladder especially when, in the case of pericholecystitis, external adhesions are formed. Independent of these different kinks are special valves inside the duct, known as Heister's valves. These folds contain muscular fibers which are specially marked where the neck of the gallbladder opens into the cystic duct, so we can speak of a sphincter in the neck of the bladder.

This sphincter muscle is of some pathological importance because a

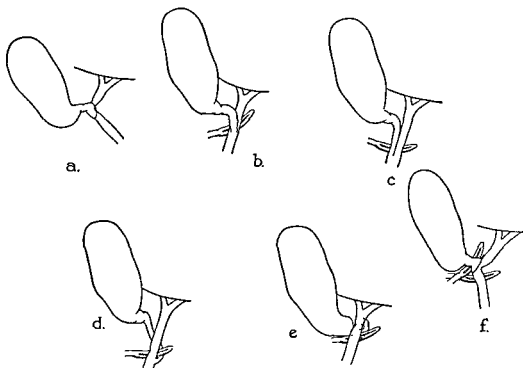


FIG. 105. Variations in the course of the cystic duct in which the common bile duct might be inadvertently ligated.

spasm around the stone lying in the neck of the bladder may cause acute and total obstruction. The diameter of the cystic duct varies considerably; the length is 2 to 4 cm. Before emptying into the common duct the cystic duct frequently forms a downward and later an upward loop in the figure of an S.

The various ways in which the cystic duct empties into the common duct are considerable and of great importance to the surgeon. The following sketches give an idea of the main variations (Fig. 104).

The abnormalities of the cystic artery are also great. It lies normally upward and behind the cystic duct, but sometimes it lies anteriorly. The surgical importance of these variations lies in the fact that under difficult conditions the common duct may easily be mistaken for the

cystic duct and be divided instead of the latter (Fig. 105). The cystic duct may practically empty into any part of the common bile duct and even directly into the papilla. So the length of the cystic duct may vary between 2 and 14 cm.

When exploring the cystic duct it is of outstanding importance not to divide the cystic duct before the anatomy is perfectly clear. Injuries to the common duct in this part of the operation are very frequent and can and must be avoided by becoming thoroughly familiar with the anatomy which is practically possible in nearly all cases. (See injuries.)

COMMON DUCT. The length of the ductus choledochus, i.e., the distance from the junction of the cystic with the hepatic duct to the papilla is between 2 and 14 cm; on an average it is 5 to 7 cm. The width of the common duct varies between 5 mm. and 2 cm. or even more.

The wall of the common bile duct consists of a mucosa, muscularis and serosa. It is usually thin, like the wall of a vein, of medium diameter, but can be considerably thickened by inflammatory processes.

In the inside of the common bile duct we find minute depressions, the function and meaning of which are still under discussion. They are considered by some authors as rudimental *vasa aberrantia*, remainders of previously functioning ducts in connection with liver tissue which has disappeared in the embryological development.

Some authors consider them real glands, while others regard these little pouches as rudimentary gallbladders.

We distinguish four parts in the common duct:

(1) supraduodenal, (2) retroduodenal, (3) pancreatic, and (4) the intramural part or the papillary part, which means the part of the common duct that pierces the duodenal wall.

The supraduodenal part reaches from the opening of the cystic duct down to the upper border of the duodenum. The retroduodenal part of the duct has on its left side the portal vein. Behind lies the trunk of the vena cava inferior. The pancreatic part of the retroduodenal common duct is the part which is imbedded in pancreatic tissue of varying degree of thickness.

Many variations are observed in the relationship between the retroduodenal part of the common duct and the pancreatic tissue. A knowledge of anatomy is important for the operation upon the retroduodenal portion of the common bile duct. The following types (Fig. 106) may be distinguished (Fuchs):

(a) The duct may lie only superficially on the pancreatic tissue. It forms a figure of S and is free from pancreatic tissue on the anterior side.

(b) The duct pierces the pancreatic tissue in a canal but is free before piercing the duodenal wall.

(c) The duct is entirely surrounded by pancreatic tissue. In order to lay bare the duct this tissue must be divided. Abundant blood vessels,

branches of varying diameter from the pancreatico-duodenal artery and tributaries of the pancreatico-duodenal vein, form the rich blood supply of this retroduodenal part of the duct.

(d) The intramural part: This part of the duct pierces in an oblique direction the posterior wall of the second part of the duodenum for a length of 1 to 1½ cm. It empties with the pancreatic duct into the so-

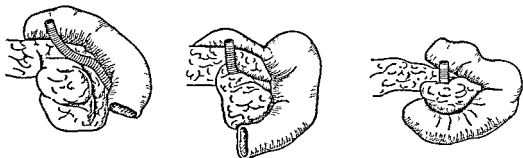


FIG. 106. Relation of the common bile duct to the pancreas I. Duct lies superficially to the pancreas. II. Duct lies in a groove of the pancreas. III. Duct is covered by pancreatic tissue.

called ampulla of Vater, the dilated part over the papilla, i.e., the opening into the duodenum. This part of the duct is surrounded by muscular fibers forming the sphincter of Oddi.

AMPULLA. There are many variations in the junction of the pancreatic duct with the common bile duct (Fig. 107). The ampulla is the place where obstructing stones are usually found. The removal of such impacted stones causes sometimes considerable difficulties. Under normal conditions there is no reflux neither of bile into the pancreatic duct nor of pancreatic juice into the common bile duct; either of these ducts has a muscular sphincter.

Under pathological conditions, especially when stones are impacted in the ampulla or when the wall has been damaged through inflammatory processes, a stasis or reflux into both ducts may occur.

The anatomical variations of the junction of both ducts are of importance when introducing a probe through the papilla from below during a transduodenal operation.

PAPILLA. The narrow and short canal, the opening of the common duct into the duodenum, is called papilla. In other words, it is the intramural portion of the common bile duct.

Longitudinal folds of the mucous membrane, closing the opening in intimate contact with the whole canal, form the inner layer. The papilla is more firmly closed by tense circular layers of muscular and elastic fibers, the sphincter of Oddi. The papilla may be forcibly dilated by probes to a width of 1 cm. It lies in about the middle of the second part of the duodenum in the posterior wall. But this topography is of no great value for finding the papilla because the distal part of the duo-

denum is covered with the transverse mesocolon. So it is of more practical value to know that the distance from the pylorus to the papilla is about 10 to 12 cm. In the open duodenum a permanent transverse fold may hint as to the position of the papilla. Near the papilla we find sometimes another small opening of the accessory pancreatic duct.

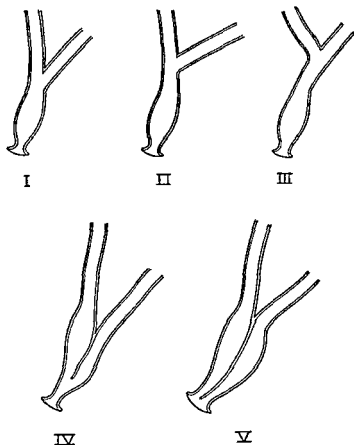


FIG 107. Variations in junction of the common bile duct with the pancreatic duct (after Waltzell). I. Both ducts empty into ampulla. II. Pancreatic duct empties into the common bile duct. III. Common bile duct empties into pancreatic duct. IV. Separation of the ducts by an incomplete septum. V. Separation of the ducts by a complete septum.

(Santorini.) This accessory duct is, when present, usually very small, of the diameter of a thread or a knitting needle and receives small branches from the head of the pancreas. This accessory duct crosses the common duct anteriorly.

ARTERIES. The hepatic artery originates from the celiac trunk (celiac artery). It is there covered by the upper border of the pancreas and goes horizontally to the right, entering the hepatoduodenal ligament near the pylorus.

It is found easily here when dividing the lesser omentum above and medial to the pylorus. This is the place of the division into the proper hepatic artery, going upward to the liver and the gastroduodenal artery going downward to the pylorus. This division lies anterior to the portal vein but is sometimes found even overlying the common duct.

The hepatic artery proper runs on the left side of the portal vein toward the liver (Fig. 102). Here we find a division into a right and left

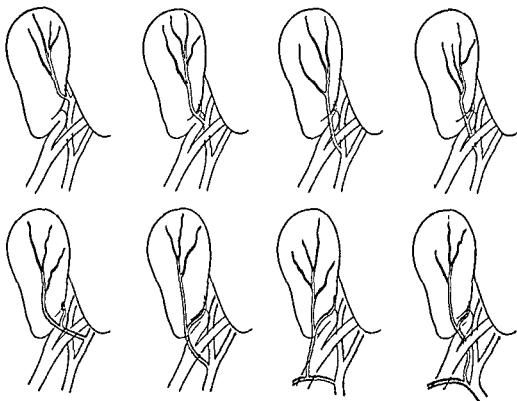


FIG. 108. Abnormalities of cystic artery.

branches. In rare cases both arteries originate separately from the common hepatic artery. From this right branch the cystic artery originates. The right branch of the hepatic artery crosses the hepatic duct on the liver hilus and supplies the right and the quadrate lobes of the liver. In rare cases the artery lies posterior to the hepatic duct and can be found sometimes even behind the cystic duct. This anomaly is also of surgical importance because it can happen that the hepatic artery may be ligated instead of the cystic artery. This accident can even be fatal, because being the only nutritive artery to the liver, ligation of the hepatic artery may be followed by an extensive ischemic necrosis.

A fortunate exception is the case in which accessory liver arteries (25 per cent) are found which originate from the left gastric artery, the superior mesenteric artery or gastroduodenal artery. These arteries are

called according to their origin the hepatomesaraic artery (Rio Branco) or hepaticogastric artery.

The *cystic artery* (Fig. 108) originates in most cases from the right hepatic artery. It shows a great number of abnormalities as to the place of its origin and especially in its course. It is found above and deeper than the cystic duct, imbedded in the hepatoduodenal ligament.

The cystic artery is always of great importance in biliary surgery. It has to be ligated in most cases of cholecystectomy, but it is useful to bear in mind the great number of various types as illustrated in the diagram.

The *portal vein* originates behind the head of the pancreas through the junction of the superior mesenteric vein and the splenic vein, whereas the inferior mesenteric vein empties in most cases into the splenic vein. It crosses the duodenum posteriorly and enters the hepatoduodenal ligament. The portal vein lies in most cases posterior to the common duct and the hepatic artery. During its course in the hepatoduodenal ligament the portal vein receives the following tributaries: the pancreatic and duodenal, the left gastric and the cystic veins.

On the hilus of the liver the portal vein divides into a right and left branches. There is in most cases still a communication between the left branch and the obliterated umbilical vein in the round ligament. The round ligament for this reason should always be divided between two ligatures. Veins of the falciform ligament also empty into the portal vein. It is further important to know, and one realizes this fact sometimes in a very unpleasant way when opening the common duct, that on its wall lies a venous plexus frequently emptying its blood into the portal vein.

The cystic vein empties sometimes directly into the portal vein. But there are venous communications of various diameters between the cystic vein and the venous plexus lying around the common duct.

Disturbing bleeding may arise out of these venous plexuses when we perform a choledochotomy, so that it is always advisable to apply ligatures and control the hemorrhage before opening.

It is further important to know that the pancreatic or duodenal vein crosses the pancreatic part of the common duct dorsally, receiving smaller veins from the pancreas and the common duct. The abundant vascularization on this place causes difficulty sometimes when operating on the retroduodenal part of the common duct.

LYMPHATIC VESSELS (Fig. 102). The lymphatic vessels of the hepatobiliary system are of special physiological and pathological interest. Inflammatory and toxic products drained from the liver and the gallbladder are collected in the glands along the cystic and common duct and into the glands along the aorta.

Lymphatic spaces lie under Glisson's capsule collecting edematous and inflammatory effusion from the liver. Inflammatory processes in

these subcapsular lymphatic vessels cause thickening of the fibrous tissues with a formation of scars (perihepatitis lymphatica). Lymphatic glands alongside the extrahepatic ducts are almost regular. One or two glands are found regularly on the left side of the neck of the bladder near the opening into the cystic duct. These glands are frequently found swollen and may cause disturbing hemorrhage when performing a subserous cholecystectomy.

To the right and to the left of the common duct glands of varying size are found; swollen glands may even cause a compression of the common duct with subsequent jaundice. Also calcified glands, apparently of tuberculous origin, can be found causing external compression. There are also communications between the lymphatic vessels of the common duct and the lymphatic glands surrounding the pancreas. These lymphatic vessels of the common duct and the lymphatic glands surrounding the pancreas form a channel of infection spreading from the hepatobiliary system to the pancreas. There are also lymphatic communications between the lymphatic vessels around the common duct and retrocecal vessels which may carry germs from an infected appendix to the hepatobiliary lymphatic system. This path of infection easily explains the coincidence of inflammatory processes in the appendix and the gallbladder.

NERVES. The nerves may be mentioned only briefly. Their clinical importance need not be explained. They carry the impulses of spastic conditions and signalize a colic attack; they regulate the co-operation between the digestion of food in the intestinal tract and the secretion and emptying of bile. Many symptoms of pathological processes in the hepatobiliary system, as perspiration, vomiting, constipation, brachy- and tachycardia, the so-called visceral reflexes, are well known. Damage through pathological processes or injuries when operating (cholecystectomy) may form the basis of the spastic or atonic dyskinesia.

The nerves of the biliary system are interrupted by ganglion cells. They originate from the vagus and the splanchnic trunk in the solar plexus. We may distinguish several different plexuses around the external biliary system: one plexus around the hepatic artery (hepatic plexus), another around the gallbladder (vesical plexus) and a third around the common duct. On both sides of the common duct two main trunks can be observed. A plexus surrounds also the sphincter of Oddi; these divisions of the plexus surrounding the common duct and the ampulla have certainly an influence on the tonus of the sphincter of Oddi. It may be that a removal of these nerves will result in a relaxation of the sphincter comparable to arterial sympathectomy, at least in some cases.

DUODENUM. It seems quite useful to mention in a few words the topography of the duodenum which is always in the field when operating upon the biliary tract. The first part of the duodenum lies posteriorly

for fourteen years used the costal incision (Figs. 110, 111, 112, 113) exclusively in more than 1,000 cases of gallstone diseases; and I am today perfectly satisfied that this incision has definite advantages; therefore, I believe I can take the responsibility of recommending it highly as a routine incision. It has proved to be a definite progress compared with the incisions formerly in use. This opinion has been confirmed by all surgeons who have tried it on a larger scale: Stegemann in 700 cases; Usadel, who recommends it as a routine incision for all gallstone operations, Mirizzi, and others.

(1) The main point of the incision is that the rectus muscle as well as the external oblique abdominal muscle are divided near the origin upon the costal arch as their basis. No damage is done to these muscles when dividing them at this place. The transverse and internal abdominal muscles are divided under the costal arch, so that both muscle incisions are on a different plane. The costal arch lying under the first muscle incision and forming the basis makes the incision safe against a post-operative incisional hernia. Not a single case of such a hernia or even a weakening in the abdominal wall could be observed in the follow-up of my cases, neither by Stegemann in his 700 cases nor in the cases of Usadel. (The frequency of incisional herniae with other incisions are given in statistical figures between 5 and 15 per cent.)

(2) The exposure is perfect so that the operation can be performed *in situ* and no displacement or traction on the liver is necessary.

(3) The opening of the abdominal cavity and the operative field is limited to the very site of the pathological changes in which a definite state of local immunity exists. Experience has shown that as long as we limit the operation to this region of immunity the tendency to heal is far better and the recovery uneventful in the majority of cases. Spreading of the infection does not occur as can be the case when extending the operative field to parts without local immunity.

The importance of this local immunity over the very site of the inflammation and the favorable tendency to heal with this incision could be shown in trials for the primary closure of the abdominal wall even in badly infected cases.

Costal Incision of Pribram (Figs. 110, 111, 112, 113)

TECHNIC. The incision starts on the right lateral border of the xiphoid process and goes downward upon the costal arch, a distance of one-fourth to one inch above the border, until it reaches the point where the ninth rib joins the costal arch; the incision is approximately eight inches. The rectus and external oblique abdominal muscle are now exposed at their origin where they are covered by the pectoral fascia; they are divided upon the costal basis and it is remarkable that practically no retraction of the muscular stump takes place.

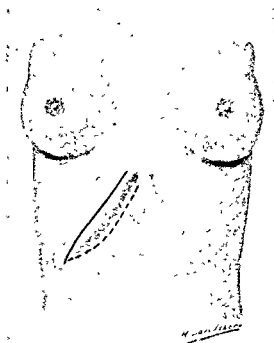


FIG. 110. Right costal incision of Pribram. Straight line — Incision through skin and superficial muscles. Dotted line --- Incision through transversus abdominis muscle and peritoneum.

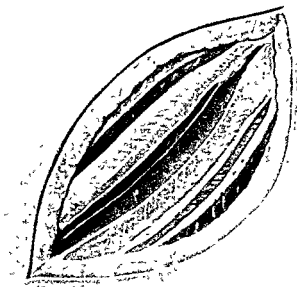


FIG. 111. Costal incision of Pribram. The skin, superficial fascia, anterior sheath of m. rectus abdominis, the m. rectus are cut and retracted.

The costal cartilage should be left uninjured. Only a few vessels have to be ligated. With two retractors the muscular stumps are now pulled medially. In the angle between the xyphoid process and the costal arch

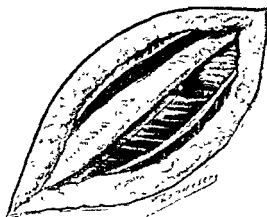
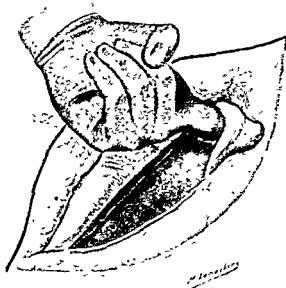


FIG. 112. Costal incision of Pribram. Dotted line shows the place of cutting of transverse abdominal muscle.

FIG. 113 Costal incision of Pribram. The abdomen is opened. Falciform ligament is lifted with the left index finger.



lie the superior epigastric artery and veins and the sixth intercostal nerve and the rectus muscle. These vessels and the nerve can be pushed aside easily.

The internal and transverse abdominal muscles are now divided a

quarter of an inch below the costal arch. Upon this muscle sheet we encounter the eighth intercostal nerve, supplying the part of the rectus muscle between the first and the second *inscriptio tendinea*.

In the beginning I have always tried to save it by pushing it aside but experience has shown that after dividing the eighth nerve at this point no weakening of the rectal muscle takes place. The round ligament is divided between two catgut ligatures which are left long so that the ligament can be tied together after the operation.

After having opened the peritoneum we are right over the gallbladder and the biliary ducts.

I will not omit this occasion to emphasize the importance of the use of a self-retaining retractor for the permanent wide exposure when operating upon the biliary tract.

The closure of the abdominal wall is performed in three layers: Continuous suture of the peritoneum with fascia transversalis and the transversus abdominis muscle. The internal oblique muscle is sutured with interrupted catgut sutures, thus closing the abdominal wound under the costal arch. The suture of the rectus and external oblique muscles is performed beginning with the epigastric end. The sutures go through the firm fascial sheet and are tied in such a way that the muscular tissue is inverted and the muscular suture is properly covered everywhere with fascia and then interrupted skin sutures. Primary closure of the abdominal wall is safe in the majority of cases especially after an electro-surgical operation.

DRAINAGE. In case drainage is necessary the drain is allowed to pass at the lower end where both incisions join in one plane. If this opening should not be suitable for drainage (as in the case of a greatly enlarged liver), the drain is allowed to pass through a special button-hole incision in a suitable place below the lower end of the incision. This special button-hole drainage has proved very useful so that it can be widely applied whenever it is suitable for the course of the drain.

WIDER EXPOSURE FOR INJURIES. When we have started with a median or paramedian incision for injuries, the amount of which could not be foreseen, we have to face sometimes the necessity for a wider exposure of the liver.

A transverse incision, perpendicular to the longitudinal incision, is carried out below the costal arch reaching the median incision about two inches below the xyphoid process. The incision goes right through the rectus muscle and as far as necessary laterally through the oblique and transverse abdominal muscles, as we approach the lower surface of the liver, gallbladder and the external bile ducts. The tendency to heal after this incision is better as one should expect; in fact, it is better than all the other incisions combined as well as the incisions below the costal arch.

Intercostal nerves supplying the rectus muscle can easily be avoided. In some cases in which a wider exposure of the convexity of the liver is required a temporary resection of the costal arch pedunculated on the intercostal muscles serves this purpose (Marwedel incision). The costal arch is laid bare by an oblique incision parallel or upon the costal arch. The muscle stumps are pushed upward so that the cartilages from the seventh rib downward are all accessible. The costocartilagenous junction of ribs is divided externally and near the sternum medially. When dividing the seventh rib, precaution is necessary lest the pleura be opened. Intercostal muscles are divided, arteries ligated and a flap pushed upward.

WIDER EXPOSURE OF THE LIVER. In special cases when operating for an injury on the convexity of the liver or for an abscess, hydatid abscess, wider exposure of the liver may be necessary. The partial mobilization of the liver by cutting the ligaments produces a useful widening of the exposure.

The posterior surface of the liver is fixed to the diaphragm without the liver being covered by peritoneum. Another and very strong fixation is given by the inferior vena cava and the hepatic vein. The division of the so-called ligaments of the liver, the round and the falciform ligament, is very useful for allowing a certain twisting around the horizontal axis. Wider mobilization may be achieved when dividing the lateral ligaments.

These ligaments are reflections of the peritoneum from the anterior abdominal wall into the diaphragm extending to the liver and blending into the capsule. These reflections are called the right triangular ligament or hepatophrenic ligament and is fixed to the right lobe on the convexity of the liver and the hepatorenal ligament, which is fixed to the lower surface and to the left coronary ligament.

When separating the convexity of the liver from the diaphragm, we must be careful not to perforate the latter. There is danger that the pleural cavity might be opened on the right side and the pericardium on the left. This mobilization can be performed only to a certain degree in order not to injure the hepatic veins and the vena cava. It should be carried out with the greatest care and under exceptional conditions, always avoiding any pulling on the hepatic vein and the vena cava which may interfere with the blood reflux to the right portion of the heart.

TEMPORARY PARALYSIS OF PHRENIC NERVE. Another kind of mobilization which has proved very useful in special cases is the temporary blocking of the right phrenic nerve by an injection of 2 to 5 cc. of a one per cent solution of novocain on the outer border of the right sternomastoid muscle, one-half to one inch above the clavicle. The following relaxation of the diaphragm enables mobilization and far better exposure. It facili-

tates, on the other hand, operating upon the liver, e.g., the performing of a proper suture.

TRANSDIAPHRAGMATIC APPROACH. The transdiaphragmatic way is by far the best for an exposure of the top of the convexity of the liver and is used especially when an operation is performed for injuries, as gunshot wounds, piercing the thoracic cavity and then going through the liver into the abdominal cavity. It is used further when an operation is performed for subphrenic abscesses. This special incision will be discussed in the chapters dealing with the technic of these operations.

Injuries to the Liver, Bile Ducts and Gallbladder

We may distinguish (1) injuries to the liver by blunt force (liver rupture) and (2) open injuries (stab wounds, gunshot wounds, war injuries).

INJURIES BY BLUNT FORCE. The size and characteristic consistency of the liver, the heaviness and ample filling with blood of the organ covered by Glisson's capsule, which has always a certain degree of tension, the fixation of the liver by ligaments to the vena cava and to the diaphragm which hinders any evading of the organ are all reasons why blunt force hitting the upper abdomen frequently causes a rupture of the liver deep into the parenchyma.

Of all parenchymatous organs in the abdominal cavity the liver is the most likely to be injured when the abdomen is hit at any part by blunt force. Street accidents, when people are run over by automobiles, falls from scaffolding, burying accidents in mines, war injuries, hoof beats, etc., are the main causes.

In 494 postmortem examinations of rupture of abdominal organs through blunt force, the liver rupture ranges in the first place with 59.9 per cent, this organ having been injured more than all other organs together (Geill). The right lobe has been found ruptured much more frequently than the left. The relationship is about 65 to 35.

MECHANISM OF THE INJURY. We may distinguish the result of a direct hit as in the case of a hoof beat in which a horseshoe-shaped rupture can be distinguished, sometimes with two deep holes of smashed liver tissue connected with an arch-shaped crack, and also the result of indirect ruptures.

We find such primary holes reaching deep into the liver parenchyma corresponding in size and form to the injuring object. In other cases, especially when the patient is run over by a car, the wheel crushes the liver over a larger area. In such cases the left lobe has sometimes been entirely divided from the right lobe through pressure exerted by the wheel against the spinal column. Railway wagon buffers may cause deep holes with star-like cracks.

Another kind of liver injury may be considered as secondary or

"contre coup" injury, in which a force hitting any part of the abdomen results in an indirect rupture of the liver.

These indirect ruptures are rather irregular because many factors determine the place and size of the rupture. It seems that these ruptures have a certain connection with the fixating ligaments. Some have a longitudinal direction along the falciform ligaments but most of them (79 out of 122, Geill) are sagittal ruptures.

In some of these sagittal ruptures it has been found that the round ligament has cut through the liver parenchyma dividing the liver into two parts. For instance, in the case of a fall from a great height the heavy weight of the liver presses down while the round ligament, fixed on the abdominal wall, holds firm. This is especially true when, as usual, the abdominal muscles become rigid.

In some cases the gallbladder has been found torn from the liver bed and a sagittal rupture occurred piercing from the bed down into the parenchyma. The holes in such a ruptured liver are found to be filled with a porridge of smashed liver tissue and with blood. This intrahepatic hematoma may be very large and being an outstanding feature had been called liver-apoplexia. These ruptures may be entirely closed and lie intrahepatically. They may be transformed into blood cysts or also into an abscess in case of a secondary infection.

It must be mentioned that, leaving aside external force, the kind of pathological state also may cause some predisposition toward a rupture. This is the case in children in whom the liver is relatively larger than in adults. At the time of digestion there is a certain amount of hyperemia and enlargement. In certain diseases (fatty degeneration, malaria, tuberculosis, syphilis) the liver is more friable.

The greatest danger of a liver rupture is hemorrhage; in fact, this is the main cause of a fatal outcome. The large intrahepatic veins have very thin walls, no valves at all, and there is no possibility to retract; therefore, the bleeding is persistent and spontaneous control is rare.

On the other hand, the hemorrhage is more likely to be venous than arterial. The amount of blood found in the abdominal cavity can sometimes be enormous. Two to three liters of blood and more have been found in the abdominal cavity not only at the postmortem examination but when operating upon patients who survived the operation.

OPEN INJURIES. The prognosis of simple stab wounds into the liver by knife or lance or even of simple gun or pistol wounds is much more favorable than in cases of a liver rupture, provided no other organs and no large vessels have been injured as well. The mortality rate is given between 10 and 20 per cent. It is well known that in case of open injuries and wounds by a missile the signs of shock, in contrast to those in liver ruptures by blunt force, may be entirely absent.

Leaving aside the sequels of an injury to a neighboring organ, the

prognosis of such a liver injury depends largely upon the amount of hemorrhage into the abdominal cavity. But there is even a possibility of a severe injury to the liver by a missile without the peritoneal cavity being opened and no blood at all is present when the abdomen is opened. This is the case when the liver is injured right under the diaphragm at the portion of its convexity which is free of peritoneum and directly adjacent to the diaphragm.

On the other hand, the pericardium or the pleural cavity can be opened easily by a shot at this point so that the blood pouring out of the liver collects in the pericardial or pleural cavity.

Sudden death after such a gunshot wound has been attributed through a "tamponade of the heart," supposed to be due to a gunshot wound of the heart. Only the postmortem examination revealed that the heart was not injured at all and the tamponade was caused by blood draining out of the wound of the liver through the injured diaphragm and the pericardium into the pericardial cavity.

The liver injury may also be undetected when blood pours through the open diaphragm into the right pleural cavity causing only chest symptoms.

I have observed several cases, when aspirating from the apparently intrapleural hemorrhage, in which the plasma of the aspirated blood appeared to be green from bile, proving that it was in fact an injury to the convexity of the liver and the diaphragm. No signs of an intraperitoneal hemorrhage were present.

Extensive injuries to the liver, especially by shell-shot wounds, have a very high mortality rate and the statistical figures from the war give a lower mortality rate (60 to 80 per cent) than actually is the case: a considerable number die on the battlefield because of immediate hemorrhage and do not reach any first aid station.

CLINICAL SIGNS. The first signs after such an injury, especially in a rupture from blunt force, are those of general shock, and under this dramatic appearance the special symptoms indicating special injury to the liver are sometimes hidden.

If we would operate upon a patient with a severe blunt injury of the abdomen as soon as the patient recovers from general shock, we would certainly have the advantage of finding the most favorable conditions for the operation when intra-abdominal organs have been injured. Of course, there is some sound objection against an exploratory laparotomy in all cases as a routine; we should try to find some diagnostic signs indicating whether or not an intra-abdominal organ has been injured.

DIFFERENTIAL DIAGNOSIS BETWEEN SYMPTOMS OF GENERAL SHOCK AND SYMPTOMS OF INJURY TO THE LIVER. In all abdominal injuries there are three groups of symptoms which are valuable in making a correct diagnosis and in determining the indications for surgical interference:

(1) symptoms of general shock; (2) general symptoms of abdominal injuries; and (3) special symptoms caused by the injured organ, in our case outflow of bile after injury to the liver.

SHOCK is characterized by the fact that practically all autonomous regulations are disturbed to a certain degree and for a certain time; regulation of peripheral circulation in the capillaries, regulation of temperature and metabolism as well as all means of biological self-defense are out of order or diminished during the time of shock.

The symptoms of shock are well known. The patient lies still with an expression of fear in his face, the skin is pale and clammy, the mind is clear but mental activity is diminished.

The respiration is shallow and frequent up to 30. Deep sighs interrupting the normal respiration are characteristic.

CIRCULATION. One of the main pathological damages in shock is that the backflow from the capillaries is seriously damaged. There is a stagnation in the capillary system; the stored blood in the spleen, liver and in the gastrointestinal tract is increased; the blood flowing to the right portion of the heart is greatly diminished and so is the amount of the circulating blood. The result is a diminished oxygenation of the tissues (anoxemia). The walls of the capillaries become damaged and the permeability increased; serum fluid transfuses into the tissue (edema) and the number of the red cells in the capillaries increases. The blood pressure is down and falls gradually according to the intensity of the shock.

These characteristic symptoms, as already mentioned, superimpose special symptoms of the abdominal injury. It is important to distinguish between these symptoms of shock and the general signs of an abdominal injury especially caused by hemorrhage.

SPECIAL SIGNS OF A LIVER INJURY. The signs of an intraperitoneal hemorrhage are outstanding in the case of a liver rupture. The general signs of loss of blood are sometimes not so striking, probably due to the fact that it is to a great extent venous bleeding from stored blood of the liver and not a hemorrhage as occurs in arterial bleeding. So the symptoms arising are influenced more by the peritoneal irritation than by the loss of blood.

When the loss of blood is considerable, the patient becomes more and more restless in contrast to the stupor he has shown in the very first moments of shock.

A dullness in the right side of the abdomen increases gradually and spreads downward, filling the whole right abdominal part. This dullness is to a certain extent shifting when the patient is turned to his left side.

Pain. Pain after a liver rupture is quite characteristic. It is specially felt when pressing the finger into the intercostal space. Pain radiates

downward and backward and also into the right shoulder, a sign of irritation of the diaphragm.

The radiation of pain into the right shoulder has been considered since the times of Celsius as a characteristic sign of liver injury, especially on its convex surface. The explanation is that Glisson's capsule and the coronary ligament are supplied by the hepatic plexus (right vagus and celiac plexus) as well as by the phrenic plexus consisting mainly of the terminal branches of the phrenic nerve. Irritation of these phrenic-nerve branches is felt not only as a diaphragmatic pain but radiates through the phrenic nerve into the cutaneous branches of the dorsal scapular nerve which originates partly with the phrenic nerve from the fourth cervical segment.

When pressing the phrenic nerve on the outer border of the right sternomastoid muscle over the clavicle, we find it definitely more tender than the left one. This phrenic nerve symptom is a very valuable diagnostic sign for lesions of the diaphragm and the convexity of the liver.

Breathing is shallow, more frequent and rather painful. But it must be borne in mind that pain as well as rigidity of muscles can be caused also by tearing of muscles and bruising in the abdominal wall.

Furthermore, fracture of the costal arch with its signs—inhibited and painful breathing—is sometimes so outstanding that the diagnosis of an injury to a special organ in the very beginning is often difficult.

Under the special symptoms of a liver injury must be mentioned:

Bradycardia. This bradycardia, which in contrast to the bradycardia produced by shock, lasts much longer over hours and days and is due to a resorption of bile out of the abdominal cavity. Bile salts have a special and definite depressing influence. This bradycardia is very significant in cases in which, considering the peritoneal irritation, we would expect a much higher frequency of pulse (**Paradoxical bradycardia**). We distinguish this bradycardia from shock-bradycardia by the fact that it sets in later, when the resorption of bile begins, and that it lasts longer.

Test of Bile in Serum and Urine. Bile dyes after liver injuries are found in the urine sometimes after a few hours. In the serum the increase of bilirubin is very marked and found early.

Diagnostic Tapping. There is not only no objection to aspirate some blood over the dullness for diagnostic purposes but I have always found it helpful and significant. We proceed in the following way:

A 20 cc. syringe armed with a fine needle is filled with about 10 cc. of 1 per cent novocain or percaine solution; the first drops are injected intracutaneously, then gradually subcutaneously infiltrating the whole track until we believe that we are over the peritoneum. By piercing the peritoneum and penetrating into the abdominal cavity, the injection is continued. So we avoid with certainty any injury to the bowel. I do not

think there is real danger of an accidental injury in this aspiration when performed in a skillful way.

The examination of the aspirated blood (microscopically and chemically) showing liver cells or bile dyes (bilirubin) may give evidence of the liver lesion. The microscopical examination may also reveal the presence of intestinal contents, *b. coli*, indicating that there is also a rupture of the bowels.

We are able to find the bile dyes in the extravasate before its appearance in the urine and before jaundiced sclera are apparent, which I have already observed eight and ten hours after the injury.

X-RAY EXAMINATION. X-ray examination has proved very valuable in diagnosing an additional injury to the bowels and should be performed as soon as possible, that is to say as soon as the general condition of the patient allows this examination.

The patient is examined when possible in his bed in an upright position under the x-ray screen. In case of a rupture of the bowel or stomach, a very characteristic crescent-shaped collection of air can be seen between the convexity of the liver and the diaphragm.

One or two x-ray pictures should always be taken to confirm the diagnosis which sometimes can be made at the first glance when screening the patient.

Pathological Development and Sequels of Liver Injuries

1. **HEMORRHAGE.** As already mentioned, hemorrhage may be fatal in a very short time and especially after gunshot wounds. The patients die from hemorrhage already on the battlefield. In other cases the bleeding takes a prolonged course owing to the low blood pressure of the intra-hepatic veins. Two and three liters of blood and even more have been found not only at the postmortem examination but even when the operation has been performed successfully. The hemorrhage may come to a spontaneous standstill and healing may take place without surgical interference.

The reflex contraction and rigidity of the abdominal muscles help the spontaneous primary control of hemorrhage and are to be considered as a sort of self-defense. When the contraction of the muscles relaxes either under morphine or under anesthesia, the bleeding from the liver may become torrential as Grey Turner rightly emphasizes.

But these recurrent hemorrhages may take place even after days when the patient has been already on the way to recovery. These recurrent hemorrhages are practically always fatal. They give a striking warning as to the unreliability of the spontaneous stop of the hemorrhage.

Such a late hemorrhage has been recorded in a case by Harris. The patient had recovered without operation and was in his eleventh day after the injury. When sitting up in bed for the first time he collapsed

suddenly and died after a few minutes. At the postmortem examination an abundant fresh hemorrhage was found.

Even on the thirty-ninth day (Nussbaum) and after eight weeks (Biernath) a recurrent hemorrhage with fatal outcome has been reported after a primary spontaneous stoppage.

I want to point out the danger of late hemorrhage in these cases in order to emphasize that a spontaneous stoppage of the hemorrhage after a liver rupture is not reliable at all and that an operative procedure in all ruptures is safer and preferable.

2. CHOLASKOS (EFFUSION OF BILE INTO THE PERITONEAL CAVITY). BILIARY PERITONITIS. The escape of bile from a ruptured liver, torn gall-bladder or a torn bile duct may result either in general or local peritonitis.

The signs of biliary peritonitis are the general ones of a peritoneal irritation, or peritonitis in a rather slow development; fever is absent in most cases, the pulse remains slow, as has already been mentioned, due to the absorption of bile products with their cardiodepressing tendency; the abdomen becomes distended, peristalsis ceases through the damage caused to the wall of the intestines, the bowels become ballooned and a paralytic ileus develops. In some cases the resulting cholemia may be followed by cramps and finally by a comatous state leading to death in a few days.

But this event is rather rare. Such an extreme toxic state is due more to a hepatic insufficiency than to pure cholemia. In the majority of cases of biliary peritonitis the course is a more chronic one with decreased tendency to resorption of bile.

There are cases reported in which several pints of bile in the abdominal cavity have been found at operation, having collected there during months without causing jaundice or signs of bile intoxication.

I had to operate upon a gunshot wound through the abdomen six weeks after the injury and found more than two liters of bile, the only sign having been a general cachexia. It seems that the degree of intoxication through bile is less marked when a slow drainage of bile into the peritoneal cavity takes place. Therefore symptoms of biliary intoxication are much more acute in cases of a rupture of the extrahepatic bile ducts or the gallbladder than in the case of the rupture of the liver.

As for the causes of death in biliary peritonitis they are due more to the aseptic peritonitis than to a supposed toxic effect of bile, especially bile acids.

This could be proved also by experimental work. When injecting bile acids even in large quantities intramuscularly or intravenously animals do not die, whereas they do die when the same amount has been injected intraperitoneally.

Another proof that the peritonitis with its shock-like increase of stored

blood and the diminished amount of circulating blood is an outstanding feature is the fact that with experimental biliary peritonitis the volume of the red blood cells increases considerably.

In cases of cholaskos or in case of a local biliary peritonitis without jaundice a fibrous and plastic transformation of the sac in which the bile was collected has been found with resorption reduced to a minimum. But in all cases a general and progressing cachexia has been found.

The diagnosis of this condition is sometimes difficult but indications for urgent surgery are always present and after opening the abdomen the true nature of the injury becomes evident.

3. **PERMANENT BILE FISTULA.** Sometimes bile fistulae with a poor tendency to heal remain after a liver injury. These may be due to a torn major hepatic duct with a cicatricial stenosis at its lower end.

The treatment in such cases will be discussed in the chapter, "Injuries to the Bile Ducts."

In some cases the cause of such a bile fistula lies only in the rigidity of the upper part of the abdominal wall, big fibrous scars connecting the ribs with the liver which hinder the retraction necessary for the healing process.

In these cases mobilization of the abdominal wall with removal of the ribs and infolding of a muscular flap into the liver wound offers good results. In case of an encapsulated collection of bile in the abdominal cavity, which is not infrequently observed, a wide opening and drainage of this cavity is necessary. Results with tapping alone are not satisfactory.

4. **INFECTION OF THE LIVER WOUND.** We can distinguish between a primary and secondary infection of the injured liver: (a) primary—in open wounds; foreign bodies, such as uniform clothes and splinters, left in the liver tissue may cause a serious infection, formation of abscess and even gas-gangrene; (b) a secondary infection may take place either from the blood stream carrying germs or from the bile ducts.

Such secondary infections are observed mainly when an intrahepatic hemorrhage containing debris of necrotic tissue is gradually transformed into an abscess. A liver abscess formed on the spot of necrotic tissue is to a certain degree comparable to a delayed abscess formed after injuries to the brain by blunt force. I mention the following case: A patient fell from the second floor and was forced to stay in bed only four days on account of his abdominal pain. Two and a half months later jaundice developed and a solitary abscess was found. (Wendel.)

Another even more striking case concerned a patient who died four years after his injury on account of an abscess perforating into the free peritoneal cavity. This formation of late abscesses after a liver injury is not very rare and according to statistical figures (Gage) develop in about 5 per cent of cases. Subphrenic abscesses following a rupture of the con-

vexity are not rare and are due to secondary infection of a subphrenic hematoma.

5. **EMBOLISM OF LIVER TISSUE.** Pieces of liver tissue of various size have been found in the lung, in the right auricle and in the pulmonary artery. Wilms found a piece weighing 21 grams in the right auricle. Fat embolism is also observed.

6. **INFARCTION OF THE LIVER.** Such infarctions may be due to a thrombotic obstruction of tributaries of the portal vein or of branches of the hepatic artery.

7. **LUNG COMPLICATIONS.** Pneumonia apparently also due to embolism is not infrequently observed.

8. **THE HEPATORENAL SYNDROME.** A number of cases have been reported in which serious renal symptoms followed a liver trauma. The main clinical signs were hematuria followed by a fatal anuria or even a primary anuria which could not be influenced by therapeutical means and which lead to a fatal issue. The kidneys showed signs of a high degree of nephrosis (lipoid degeneration); the cells in the tubuli were found to be without nuclei and in a state of degeneration and necrosis.

It is remarkable that the epithelium of the *tubuli contorti* is found to be more damaged than the glomeruli which are sometimes found not damaged at all. Apparently some potent toxic substances are reabsorbed from the necrotic liver cells and excreted by the kidneys causing there considerable damage to the convoluted tubuli.

9. **EYE SYMPTOMS.** Changes in the fundus of the eyes have been observed after a liver rupture. These consist in hemorrhage and disturbances of circulation in the optic nerve leading in some cases to transitory amaurosis. In some cases a picture like in albuminuric retinitis results.

INDICATIONS. *Strict and fast rules cannot be given when to operate for injuries of the liver or biliary passages. Lack of facilities, or of a competent surgeon, which prevents an immediate operation, may considerably change the indications for an urgent operation.*

I think it is more advisable to state the indications here strictly from a purely medical point of view. We shall suppose that an experienced surgeon has to deal with the case early after the injury in a modern clinic with all assistance at hand, that is to say, under ideal conditions, and we shall leave it to the personal judgment of a surgeon to make the concessions necessary because of the unfavorable lack of facilities.

The main indications in liver injuries arise from questions such as: (a) Whether we think that the hemorrhage is ceasing by itself; (b) whether the external bile ducts are also injured; (c) whether there is a suspicion of other organs (intestines) being injured as well. A large amount of bile in the urine and a quickly increasing jaundice point always to an injury of the external bile ducts.

No doubt minor injuries to the liver may heal spontaneously. It is also quite certain and confirmed in many cases that gunshot wounds through the liver with rifle or machine gun bullets may heal uneventfully. But that serious ruptures also may heal without interference is shown in the following case which I just mention in passing:

A man, run down by a lorry, was admitted to the hospital with definite signs of a rupture of the liver. He had also a fracture of the skull and several fractures of vertebrae. The general state was so poor and rather hopeless that the surgeon could not make up his mind to operate at all, despite the fact that the peritoneal symptoms and the dullness in the right upper abdomen clearly showed to be due to severe hemorrhage of the liver. Only morphine was given to relieve the patient's pain. Surprisingly enough the patient was better the next day and the surgeon did not operate because the peritoneal symptoms were decreasing and the hemorrhage had obviously come to a standstill. In doing so he was again certainly right. The patient recovered fully.

But this case is by no means an example of how one is to proceed as a rule and we should not rely too much upon spontaneous healing.

I am rather inclined to say that I would feel safer when in every case in which an intra-abdominal organ is supposed to be torn a laparotomy would be performed as soon as the signs of general shock have disappeared, so that we can pass a better judgment as to the character and amount of injury.

We are sometimes surprised to see injuries, like a torn mesentery, etc., of an unexpected kind which we can repair very easily and which certainly would have caused a fatal outcome without operation.

That the liver may be injured to a large extent and that nevertheless full recovery may take place are shown by the following interesting case reported by Branch:

A young boy, seven years of age, had been struck by an automobile, the front and rear wheels of one side passing over his body. When operating upon him, it was found that the entire left lobe and a portion of the right lobe of the liver had become nearly completely separated. This tear ran from the point of entry of the round ligament posteriorly into the right so that practically one-half of the liver substance was lying free. This part of the liver was doubly clamped and removed. The weight was 354 grams and measured 17 by 9 by 6 cm. A mattress suture was placed, closing the raw surface of the liver and a gauze drain introduced. A bile-stained drainage took place for a fortnight, otherwise the recovery was quite uneventful. When bearing in mind that the liver weight of a male of this age is about 680 grams, it can be said that one-half of the liver substance was lost.

Obviously employment of liver sutures cannot be carried out under unfavorable conditions, especially in war surgery.

According to statistical figures in non-operated cases of liver ruptures the mortality rate is up to 85.7 per cent (189 cases, Edler). Out of thirteen cases of liver rupture operated upon by Terrier and Auvray the mortality rate was 53.85 per cent and later out of ten ruptures 30 per cent.

In order to compare the improvement of liver injuries as a whole when operated upon in due time with the statistical figures in non-operated cases, we must resort to earlier statistics because we can rarely find today statistics of nonoperated cases.

The total mortality rate in 543 nonoperated cases was found to be 66.8 per cent, while in operated cases the mortality rate dropped to 28.57 per cent. From all the statistical figures it has been evident that ruptures have a far higher mortality than stab and simple gunshot wounds. But a great deal depends on whether or not neighboring organs, large blood vessels, pancreas and intestinal loops are injured as well.

The high mortality of course is due to infected shellshot wounds, ploughing up of the whole right side, to presence of foreign bodies such as particles of clothes, broken ribs, which are infected, and to splinters, piercing deep into the liver parenchyma.

When operated upon in due time the results of treatment could be improved considerably. According to Edler's statistics in case of stab wounds the mortality dropped from 64.6 to 24.65 per cent, in gunshot wounds from 49 to 35 per cent and in case of liver ruptures from 85.7 to 61.4 per cent. As for the causes of death, loss of blood ranges in the first place, followed by other complications, like peritonitis and other secondary sequels of the primary injury.

Many surgeons have more conservative indications concerning liver injuries. I quote G. Gordon Taylor: "If injury to other viscera can be excluded, abstention is the best policy."

It must be admitted that in the great strain which sometimes exists in war surgery when hundreds and hundreds have to be treated in the shortest time, indications of abdominal surgery as a whole are bound to become more conservative.

But, as stated before, my purpose here is to give indications from a purely medical point of view, irrespective of external conditions, and to leave it to the judgment of the surgeon to make the necessary concessions demanded by existing facilities. As to the leading point in Gordon Taylor's indications, namely, that abstention is the best policy if injuries of other viscera can be excluded, it must be said that this is not often the case. Contre-coup injuries of viscera may take place in an absolutely unexpected way. I remember a case with a tangential shot smashing a few ribs and injuring slightly the outer border of the liver which was found scarcely bleeding. But laparotomy revealed a large amount of blood in the abdominal cavity because of a torn mesentery.

IN SUMMARIZING the reasons why in all cases of injuries to the liver either by blunt force or by gunshot wounds laparotomy as an urgent operation should be carried out, provided there are adequate facilities for their execution, I would say as follows:

(1) In most cases there is no certain evidence that the liver is the only organ which has been injured.

(2) While a spontaneous arrest of hemorrhage in minor liver injuries is quite common, there is no assurance whatever that a serious and even fatal hemorrhage may not take place again even after days of a seemingly good recovery.

(3) Secondary infection of intrahepatic hematomas or subphrenic hematomas is not uncommon even after a long time after the injury.

(4) A repair of torn bile ducts is much easier to perform shortly after the injury than after a certain time has elapsed.

Statistical figures have proved conclusively that the mortality rate in liver injuries is about one-half when operating instead of running the risk of waiting for the patient's chance of a spontaneous recovery.

Even very serious cases of war injuries, which have been given little chance of recovery at the first sight, could be saved by a well planned operation.

As in all abdominal injuries, indications and the extension of the operative procedure change when several days have elapsed from the time of injury. Indications for operation in such a late state must be considered very carefully and must meet only the urgent demand of special cases and never be a general laparotomy with the examination of all abdominal organs. It has always proved a bad procedure to perform a general operative review of the abdominal cavity in a stage when the self-defense has already taken care to localize the intra-abdominal process.

Operative Procedures

In all abdominal injuries we have first to deal with the symptoms of shock, the first rule being that we should never operate upon a patient who is in shock.

TREATMENT OF SHOCK. The general disorder in the whole autonomic nervous system, the damage done to the nerve regulations of all organs and vital functions, especially the lack of any self-defense during shock, explain why a hastened operation is dangerous. The patient may die in sudden collapse at the very beginning when general anesthesia has been started. We can even run the risk of increased peritoneal hemorrhage rather than that of an operation in shock. The preoperative treatment is, therefore, in all these cases a treatment of shock in the first place. This subject of treatment of shock is treated elsewhere to which I refer the reader.

ANESTHESIA. I always favor spinal anesthesia for its complete relaxation of the abdominal wall which allows a quick examination of all intra-abdominal organs. In addition, gas oxygen is useful to maintain regularity of respiration. Low blood pressure is a contraindication to spinal anesthesia. In these cases only general anesthesia with gas-oxygen-ether should be given; simple local anesthesia should be avoided. There is no advantage but serious disadvantage in it (increase of shock, restlessness, insufficient exposure).

INCISION. In all injuries to the liver, whether caused by blunt force or in the case of penetrating wounds, we must bear in mind that other organs may be injured as well. Therefore, I think that in all these cases a nonspecific incision, like the median or paramedian, offers a general exposure of all the organs of the abdominal cavity and is most satisfactory. An additional right angular incision in order to approach the liver may be helpful. In special cases, when the rupture lies high up in the convexity, a special and wider exposure of the liver may be necessary; the median explorative incision may be closed and one of the special incisions with wider exposure of the liver described previously should be performed.

In an open injury of the abdomen (stab-wound or gunshot-wound), it is always advisable to explore first the abdominal wall through the already formed channel after the edges of the wound had been carefully cleansed.

The advantage of this procedure is that we can easily see the trace of the missile or knife. Sometimes we may find a missile or shell splinter very deep in the abdominal muscles without the peritoneum having been opened at all and the whole laparotomy may be spared. These cases in which a missile pierces only the abdominal wall and is found in a distant part of the abdominal muscles are well known. When opening the peritoneum through the place of the wound, we may have a fairly good view of the organs injured and can enlarge the incision in any suitable direction.

After the first inspection and when the plan of the operation has been conceived, the primary wound may be closed and the abdominal cavity explored by one of the routine incisions.

After dividing the peritoneum a large amount of blood pours out which should be removed at once by suction or by large abdominal pads. At times the bleeding is profuse and apparently starts with increased violence when by opening the peritoneum the intra-abdominal counterpressure relaxes. Quick acting is necessary, otherwise the patient may collapse and die in a few minutes. The main source of bleeding must be found immediately. Temporary compression of the aorta right under the diaphragm exerted by the assistant is the best help in such cases.

When all other common sources of such hemorrhages (torn abdominal vessels, spleen, etc.) can be excluded and the liver is found to be the only source of the bleeding, compression of the aorta can be replaced by compression of the hepatoduodenal ligament (J. Hogarth Pringle, Tuffier, etc.)

This can be done by the help of the fingers or with a soft clamp, the branches of which are covered with rubber. Spivack's clamp is very convenient for this purpose. Within a few minutes the liver has emptied a great deal of its blood into the right heart and the bleeding from the wound in the liver stops. But this compression should not be carried out any longer than five minutes and an assistant should observe the conditions of heart and circulation carefully.

The procedure is not without danger. While the blood supply of the hepatic artery can be checked for a longer time without damage, the obstruction of the portal vein may result in a serious drop of blood pressure. There is also a certain danger of congestion in the mesenteric veins and in the intestines.

It has proved useful in cases in which the portal vein has to be compressed to compress as well the aorta under the diaphragm. It has been observed that in this way the fall of the blood pressure following compression of the portal vein can be prevented to a certain degree by checking the emptying of the left heart.

When the place of rupture in the liver is exposed, the operative field is walled off with large abdominal pads moistened in hot saline. We have to satisfy ourselves that no bile ducts are torn. The ruptured part of the liver is provisionally plugged with gauze and then the final control performed while the gauze is gradually removed. All our aims must be directed to the sparing of as much blood as possible.

LIVER SUTURE. Ligature of bleeding vessels in the wounded surface of the liver is sometimes quite satisfactory. A suture is performed through the liver tissue around the bleeding vessels in a purse-string way.

In large and deep ruptures the control of hemorrhage may be useful as is described in the case of liver resection. This procedure is based on the fact that the walls of blood vessels are of greater resistance than liver tissue, so when tightening the suture the threads cut through the crushed liver tissue but do not cut through the intrahepatic vessels which can be properly closed by ligature. The procedure (Fig. 114) is as follows (Kousnetzoff and Pensky):

A long, flat, blunt needle threaded with a double thread of catgut No. 5 is pierced through the liver near the border. One of these threads is cut near the eye and the first ligature performed with this free thread. In this way the whole bleeding surface of the liver is checked by such sutures.

If a needle with a dull point should not be available, as may happen in urgent surgery, an ordinary needle may be used with the threaded eye directed forward. The suture must be tied slowly and very gently.

A useful modification of this method has been developed by Auvray. The suture is performed by means of a Reverdin needle (Fig. 115).

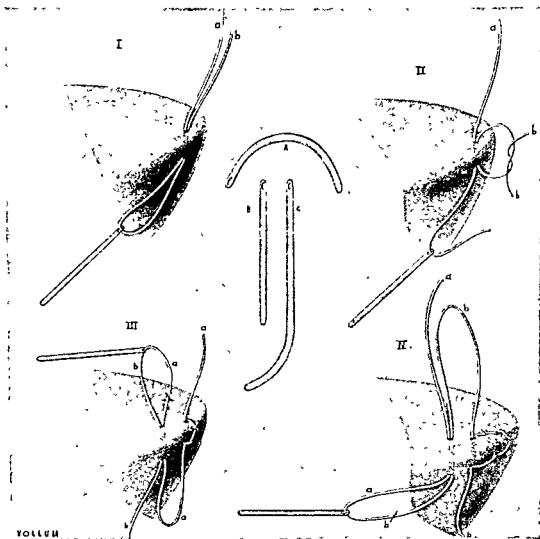


FIG. 114. Suturing of the liver (Kousnetzoff-Pensky method). (From Julius L. Spivack's "The Surgical Technic of Abdominal Operations," 4th edition 1946. Courtesy of Charles C Thomas, Publisher, Springfield, Illinois)

In all these deep sutures there is the danger of a partial necrosis of liver tissue. But it must be said that in injuries we are rarely forced to apply one of these rather complicated methods which are more suitable as prophylactic methods of blood control before partial resection of the liver. We succeed in most injuries with one of the following methods of direct suture with or without a free graft tissue.

In some cases the suture of the liver can be performed without serious difficulties provided a big needle with large size catgut is used.

The consistency of the liver tissue varies considerably. It is sometimes so friable, especially in cases of fatty degeneration, that all sutures cut

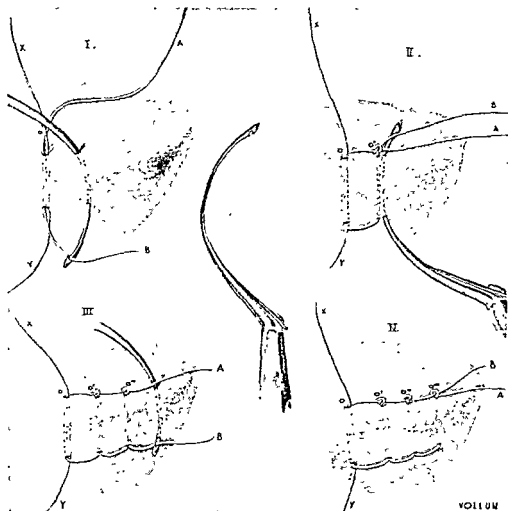


FIG. 115. Suturing of the liver (Auvray method). (From Julius L. Spivack's "The Surgical Technic of Abdominal Operations," 4th edition 1946 Courtesy of Charles C Thomas, Publisher, Springfield, Illinois.)

through and the bleeding increases during the suturing instead of being stopped. In these cases a different technic must be applied.

1. **MATTRESS SUTURES** are applied in such a way that the different threads cross each other. The main thing is that the mattress sutures are put in an oblique direction instead of a rectangular one so that each suture is crossed by the following suture. The sutures must go deep down into the liver tissue. Otherwise, when only suturing both surfaces,

a gap remains in the middle and the bleeding continues to pour out between the sutures (Fig. 116).

2. Another method is to introduce one or two long, thick catgut sutures parallel to the wounded surface; they are laid at a distance of 1 or 2 inches from the wound. Through these holding sutures simple rectangular or mattress sutures are laid (Fig. 117). We shall see almost

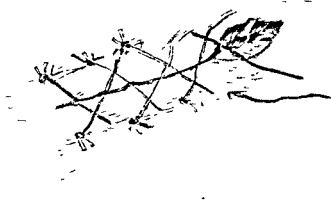


FIG. 116. Crossing liver sutures.

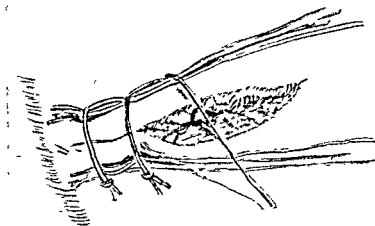


FIG. 117. Suturing of the liver by sutures over catgut.

after the first suture whether we can succeed with this technic or whether the sutures cut through the liver tissue. If such is the case another method must be applied, i.e., the method of free graft tissue:

(a) The assistant brings both parts of the ruptured liver close together, a free graft stump of fascia is laid over the wound and then simple or mattress sutures (Kirschner) are applied through the fascia. This method gives satisfactory results (Fig. 118).

(b) Both wounded surfaces of the liver are entirely covered with free

graft—omentum or fascia. The liver suture is then employed. In smaller wounds the liver suture can be performed over some free graft muscular tissue, the latter being the best to stop the hemorrhage.

The choice between these methods briefly described here is easy for the surgeon according to the individual case.

When applying one of these technics we are almost always in a position to control hemorrhage properly.

These methods of suturing, however, can rarely be performed in extensive war injuries, especially in gunshot wounds.

We sometimes find liver tissue deep in the wound apparently doomed



FIG. 118. Mattress suture for suturing the liver

to necrose and pedunculated only on a small stalk of tissue. This should not be removed without proper ligation, otherwise serious hemorrhage may follow because such a torn liver tissue may be pedunculated on larger vessels. Thorough packing is the only method which can be applied in such cases. However, we should not proceed with it before having ligated all larger vessels in the bleeding cavity. This can be done by deep sutures around the vessels.

When performing proper plugging of a large liver wound, it must be borne in mind that sometimes it is difficult to retain the plugging gauze in a soft hole of the wounded liver; but firm pressure is essential, otherwise the bleeding continues under the gauze and the purpose is not achieved. The best way in such cases is to introduce several long catgut threads from one side of a liver wound deep down through the hole and to allow the threads to pass on the other side of the surface when the plugging is performed and the sutures tied over the gauze.

There is no difficulty in gradually removing the plugs when using a Mikulicz tampon which has proved a very useful method. The wound is thoroughly lined with a single layer of gauze and filled with several gauze pads (Fig. 119).

The whole packing is allowed to pass like a bundle through the drain-

age opening. The idea is that these pads can later on be removed one after the other allowing a gradual closing of the cavity.

The gauze with which the wound is lined can be soaked with an antiseptic solution or some powder. Grey Turner advocates the dusting of the packing with boric powder which gives satisfactory results

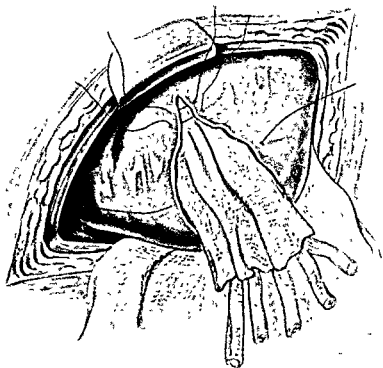


FIG. 119. Suture of the liver over Mikulicz tampon.

through its antiseptic property and some little protection against secondary infection.

Vioform gauze or gauze soaked with ferrum sesquichloride is helpful sometimes to control the hemorrhage. In infected wounds I found it a good method to soak the gauze in peruvian balsam. It has the advantage of preventing too firm adherence which may cause a secondary hemorrhage when removing the packing. On the other hand peruvian balsam is, beside being an antiseptic, also an excellent stimulant for regeneration.

TECHNIC OF REMOVAL OF THE PACKING. Only the external dressing is changed when it is soaked with blood and bile, otherwise the whole wound is left untouched for the next few days. No packing should be removed for at least eight or ten days even when we assume that an elevated temperature may be caused by the packing. Secondary hemor-

rhage is always a serious and may even be a fatal accident while removing a packing too early.

Another point is that the gauze pads must first be loosened and then very gently and gradually removed one after the other. This is the great advantage of a Mikulicz tampon.

By far the best method for loosening gauze packs has been the injection of a strong peroxide solution into the gauze. The solution not only moistens the gauze but the small oxygen bubbles also widen the mesh of the gauze and separate it from the tissue. The time spent on the removal of a larger packing should be at least three to four days. No harm can be done when delaying the removal but much harm can be done when hastening it impatiently.

We often find large necrotic pieces of liver tissue loose in the wound, but the tendency to recover is sometimes surprising considering the extent of the injury.

REINFUSION OF BLOOD COLLECTED IN THE ABDOMINAL CAVITY AFTER RUPTURE OF AN ORGAN. Reinfusion in anemic patients has been advocated by many surgeons especially in the case of a rupture of an ectopic pregnancy and it has given satisfactory results. The main condition for the suitability of this blood is, of course, that it be absolutely sterile. This is not certain in a liver rupture and in injuries to the abdomen. Furthermore, the blood is mixed with bile. On the other hand the scheme of modern blood transfusion is practically everywhere so well organized by living donors or by the means of stored blood, that I do not see any advantage in using this blood collected in the abdominal cavity. I think this procedure should be reserved only for exceptional cases: great urgency and absence of other means of blood transfusion.

When hemorrhage in a ruptured liver is thoroughly controlled, as much as possible of the blood collected in the abdominal cavity is sucked up and clots removed mechanically by means of the hands and swabs. When a liver wound has been sutured properly, the abdomen can be closed without drainage. When packing has been done the free end of the tampon gauze is brought outside the abdominal cavity through any suitable place. In a median incision it is better, in order to avoid postoperative herniae, to pass the tampon through a fresh incision on the right side of the patient.

According to the general state of the patient a blood transfusion or a driplet infusion for several days should follow as postoperative treatment.

PROCEDURE IN COMBINED GUNSHOT WOUNDS OF CHEST AND LIVER. According to statistical figures (Wieting) 15 per cent of gunshot wounds in the upper abdomen pierce through the cavity of the chest at the same time. Many of these patients die already on the battlefield from shock, hemopneumothorax and hemorrhage. But when such patients survive

the first critical hours and can be operated upon in not too hopeless a general state, there is still a chance to save their lives. Increased experience and the advance of our technic have contributed toward the improvement of operative results.

HOW TO PROCEED IN THESE CASES AND THE BEST WAY OF EXPOSURE. The following purposes must be achieved: (1) hemorrhage must be controlled; (2) the pleural cavity must be closed from the abdominal cavity; and (3) the pleural cavity must be closed so that no open pneumothorax remains.

Many of these cases, especially when a missile has crossed from the thoracic inlet piercing deeply into the abdominal outlet and injuring mesenteric vessels and intestinal loops, are rather hopeless; the majority of them die before reaching any hospital or first aid station.

The question of how to start arises: Thoracotomy first or laparotomy?

The best solution is to be guided by the most urgent symptoms. An open pneumothorax needs our aid first of all. The first stage of the operation consists in closing the cavity of the thorax from outside and from the cavity of the abdomen. Both purposes can be achieved by stitching the diaphragm to the thoracic wall.

TECHNIC OF THORACO-ABDOMINAL OPERATION FOR GUNSHOT WOUNDS. The operation has been performed by many surgeons with increasing success in the last and in the present World Wars.

Morphine is given (0.02 Gm.) and 0.001 Gm. of atropin; blood transfusion is carried out when necessary. The right phrenic nerve is temporarily paralyzed by injecting 5 cc of 1 per cent novocain solution. This is carried out in the following way:

The head of the patient is turned to the left side; the outer border of the right sternomastoid muscle is felt and pushed medially with forefinger and middlefinger of the left hand, the middlefinger lying on the sternal part of the clavicle. The point of the forefinger indicates the place where the injection is carried out; the phrenic nerve lies here under the sternomastoid muscle upon the scalenus. The following relaxation of the diaphragm is of the greatest use for the transdiaphragmatic way and for any operation on the diaphragm.

The patient is on the table in an upright position with the right arm lifted; general anesthesia is always preferable especially because pressure can be exerted on inspiration and the lung blown up when required. The shock, when opening the thorax, is much less than under local anesthesia.

The incision is made along the sixth or seventh intercostal space and broken ribs are removed. (Figs. 120, 121). We sometimes see serious hemorrhage from a hidden intercostal artery which erroneously was considered to be a hemorrhage from the liver. Removal of these pieces of ribs and proper ligature is the safest method. Ligature around the rib

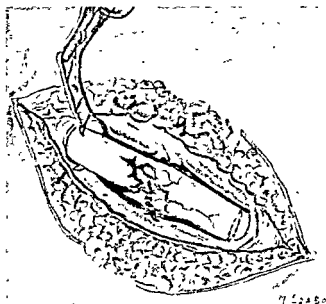


FIG. 120. Transpleural exposure. Gun shot wound of the liver.

to stop hemorrhage, as has been advocated, even with the aid of a gauze compression is by no means safe.

Bleeding wounds in the lung are sewn up, then the hole in the diaphragm is incised to explore the convexity of the liver. The diaphragm is stitched up with the pleura in order to shut off the pleural from the abdominal cavity (Fig. 122).

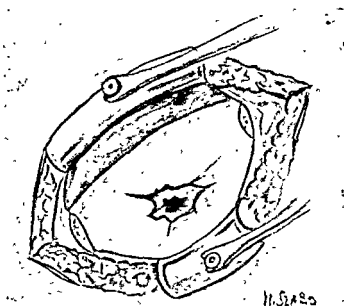


FIG. 121. Transpleural exposure. Gun shot wound of the liver.

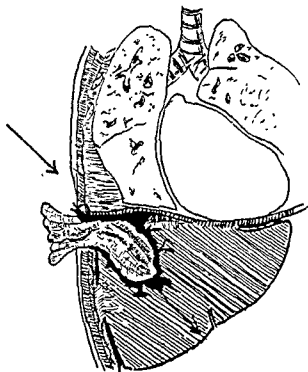


FIG. 122. Chest-liver penetrating shot wound. Chest cavity separated from the abdominal cavity by suturing the diaphragm to the abdominal wall. Closure of the chest wound. Blood from the closed haemothorax later to be aspirated.

The wound in the liver must be thoroughly explored especially in the case of shell-shot wounds and missiles and pieces of clothes and other foreign bodies carefully removed (Fig. 123). Serious suppuration may

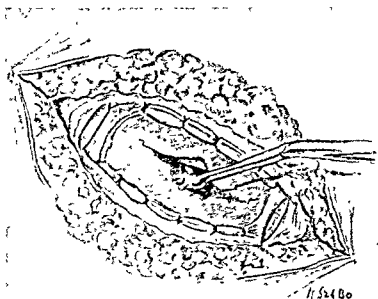


FIG. 123. Removal of a foreign body from the liver by transpleural route.

follow when infected bodies are left and even gas gangrene has been observed, the glycogen of the liver cells being a good medium for the growth of the anaerobic germs.

In most cases there can be no question of a real liver suture and plugging seems to be the only method which can be applied.

It is advisable to stitch the diaphragm together and to allow a drainage of the liver wound through an additional incision. Permanent suction through a small drain introduced in a separate intercostal space is advisable.

When this thoracic part of the operation is finished, we must decide whether an additional laparotomy is necessary. All depends on the general state of the patient. It is sometimes wise to interrupt the operation at this stage and to perform a blood transfusion. I have several times performed a laparotomy even four hours later under spinal anesthesia when the patient has recovered from the transthoracic operation.

In cases in which we think that all can be done by laparotomy, we start with the laparotomy under spinal anesthesia and expose the liver, according to the methods already mentioned. (See Anatomy.)

The blood in the pleural cavity can be removed later by aspiration.

I quote an example of how a wounded soldier, apparently in a desperate condition, can stand the strain of a very extensive operation and recover. This is the case observed by A. L. Lockwood of Toronto, and cited by G. Gordon-Taylor:

C. Gunner, age 31 years, on arrival at the casualty clearing station on September 27, 1917, was in a moribund state. He was given active resuscitation; he was placed in a heated bed and sleep was induced with pantopon; 20 ounces of a 2 per cent sodium bicarbonate solution were given intravenously. The patient rallied very slowly and he was given a 600 cc. transfusion of blood.

His condition improved after the blood transfusion and he was taken to the operating room six hours after admission. His pulse rate was 130 to 140; blood pressure, 85; respiration, rapid. The wound of entry was large and admitted three fingers; it was not a sucking wound and was situated just to the right of the body of the third lumbar vertebra. There was a dirty, jagged wound of exit admitting three fingers over the costal cartilage of the ninth rib in the anterior axillary line. Through the latter wound there was a hernia of the colon presenting a gangrenous patch the size of a quarter with a half inch laceration.

Local and paravertebral anesthesia were administered. An incision was made from the side of the third lumbar vertebra to the cartilage of the ninth rib. Excision of entry and exit wounds with the deep muscles of the back was carried out en masse. The gangrenous patch in the bowel was excised and closed with linen thread and the colon replaced. The

chest was completely filled with clotted blood. There was a wide laceration of the diaphragm five inches long and one-quarter to one-half inch from its parietal attachment. The lung was almost completely collapsed. The pleura was cleansed and the diaphragm sutured with catgut. A laceration of the outer and lower border of the liver admitted three fingers but the liver was not bleeding. The peritoneum was swabbed dry and closed. The right kidney was split across beyond repair so nephrectomy was performed. The operation was carried out entirely with paravertebral and local anesthesia; no gas and oxygen were used. The patient's recovery was uneventful and he left the hospital on November 10, 1917.

INJURIES TO THE BILE DUCTS. Isolated injuries to the external bile ducts and to the gallbladder through external force are rarely observed. In most cases it is an additional injury to the rupture of the liver. A severe crushing accident is an important cause but rupture occurs also following only a slight force.

Symptoms are pronounced and localize in the right upper abdomen when the primary shock is over. But there are cases in which abdominal symptoms appear rather late, say after twelve hours. It is interesting to note that sometimes fat necrosis has been observed without any lesion of the pancreas apparently due only to the activity of bile.

The gallbladder may be found ruptured or pierced by a missile; it may be torn from the liver bed which is found to bleed considerably; in rare cases the common duct is torn. In all these cases, the amount of bile drained into the abdominal cavity is considerably higher than after a rupture of the liver. The signs of biliary peritonitis develop quickly; jaundice is already observed after a few hours.

SURGICAL INJURIES TO THE BILE DUCTS. Most of the injuries to the bile ducts take place when a cholecystectomy for gallstones is performed. This mishap occurs mostly when operating under difficult conditions and when the inflammatory process obscures the anatomy; in other cases it is due to anatomical abnormalities which are rather frequent in this region.

The diagram (Fig. 105) shows some stages of operation in which injuries may occur:

- (1) When one pulls on the cystic duct or on the gallbladder, the wall of the common duct may come into the ligature so that a stenosis or total obstruction results.

- (2) The anatomical anomalies shown in the diagram easily explain why such injuries may take place. In one case a single hepatic duct emptied directly into the bladder with one single common duct as outlet; obviously when removing the gallbladder the dividing of the main duct was inevitable (Fig. 124).

(3) In the case of common duct stones and partial necrosis of the wall due to impacted stones and inflammation, the wall can become friable so that it may easily be torn when working there.

(4) In duodenal ulcers the common duct may be imbedded in firm scars, so that when mobilizing the duodenum for resection, the common duct may be ligatured and divided in its supraduodenal or retroduodenal part.

In order to avoid such surgical injuries some rules can be established:

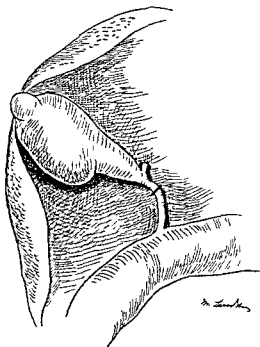


FIG. 124. Direct opening of hepatic duct into the gallbladder.

(1) The laying bare of the cystic duct should always be performed very carefully and the common duct as well as the hepatic duct laid bare. The ligature around the cystic duct near the bladder should be put on first and no clamp should be used for the occlusion of the proximal end of the cystic duct. The procedure of clamping the cystic duct, being one of the main sources of injuries to the common duct, should be strictly condemned.

(2) The wall of the common duct may be easily grasped between the blades of a forceps and tied or stenosed by the ligature. In other cases the wall may be damaged and become necrotic after a few days. The cystic duct should be divided between two ligatures not too near to the common duct. Anatomical abnormalities must be carefully considered in every special case.

(3) Before performing the exact peritonization and closing of the

abdominal wall we must satisfy ourselves that the common duct is not injured and the outflow of bile from the liver is secured.

TREATMENT OF INJURIES. Most of these injuries could be avoided even in case of rare abnormalities. When such a mishap occurs, it is of the greatest importance that it be noticed at once. Then the repair of the damage is not too difficult; at any rate, it will be much easier than at a second operation.

In such re-operations the repair may be extremely difficult because of the formation of scars which may lead sometimes to an obliteration of the common duct. It must be stated that in many instances, unfortunately, the injury is not observed during the first operation and is diagnosed only by the consequences which demand a second operation for repair of the damage.

While in injuries brought about by external force or accident the increasing symptoms of general peritonitis are strict indications for immediate operation, the symptoms arising from a surgical injury—mainly a ligature of the hepatic duct—are not very striking at first. Jaundice sets in gradually but the general conditions may not appear to be serious. This state may last for months before a second operation is decided upon due to the gradually deteriorating symptoms.

The stasis of bile above the obstruction may cause enormous dilatation of the intrahepatic bile ducts leading even to intrahepatic rupture and to perforation through the diaphragm into the lung and into the bronchi.

Such a case was reported recently by Douglas Miller:

A man, 52 years of age, was operated upon several times for attacks in the right hypochondrium: gastroenterostomy, cholecystectomy and two years previously cholecystostomy have been performed. The last operation, in which the hepatic duct apparently had been ligatured, was followed by very intense pain and jaundice. One and a half years after the last operation he coughed out a large quantity of bile and since then up to 60 ounces of bile a day.

Operation and postmortem examination revealed a cicatricial obstruction of the hepatic duct at the porta hepatis. Proximal to this the ducts were enormously dilated; a small fistulous track led from one of these dilated ducts to the upper surface of the liver and then through the diaphragm into the lung.

REPAIR OF BILE DUCT INJURIES. The procedure depends mainly upon the place of the injury and upon the part of the undamaged duct that is left. We may distinguish between the following operations: (1) direct suture; (2) implantations; (3) side-tracking operations, i.e., cholecystoduodenostomy; and (4) plastic operations. The following diagram presents a scheme of operations for repair (Fig. 125). We may summarize the idea of the plan as follows:

- I. Injuries of the cystic duct
Repair: cholecystectomy
- II. Common duct torn in its supra- or retroduodenal part
Repair:
 - (a) direct suture over a tube
 - (b) ligation of the distal end and choledochoduodenostomy
 - (c) ligation of both ends, cholecystoduodenostomy or cholecystogastrostomy.

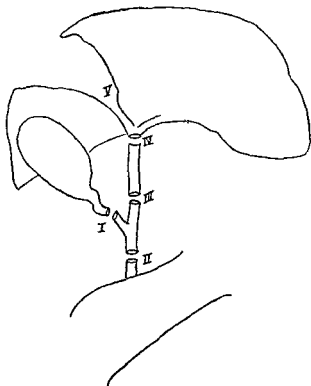


FIG. 125 Schema of injuries to the bile ducts and their repair. I. Cystic duct is torn: Repair: Cholecystectomy. II. Common duct is divided near the duodenum: Repair: (a) direct suture over rubber tube, (b) Ligature plus cholecystoduodenostomy, (c) Ligature plus choledochoduodenostomy. III. Hepatic duct is divided in the middle: Repair: direct suture with or without aid of rubber tube. IV. Hepatic duct is divided next to the liver. Repair: plastic operation: (a) Implantation over rubber-tube into the stomach or duodenum, (b) Plastic method after Walton-Moynihan. V. Intrahepatic injury of main hepatic duct: Repair: (a) Drainage with rubber tube through the common duct into the duodenum, (b) Packing of the liver wound.

III. Injury of the hepatic duct

Repair:

- (a) proximal stump long enough and suitable: direct end-to-end suture of the divided ends
- (b) proximal stump too short and unsuitable:

Repair: plastic operation over a rubber tube

(a) cholangioenterostomy

(b) hepaticoenterostomy

IV. Injury of big intrahepatic duct (liver rupture)

Repair: drainage into common duct

TREATMENT OF INJURIES. The best way to secure a safe outflow of bile from the liver into the duodenum is always the reconstruction and

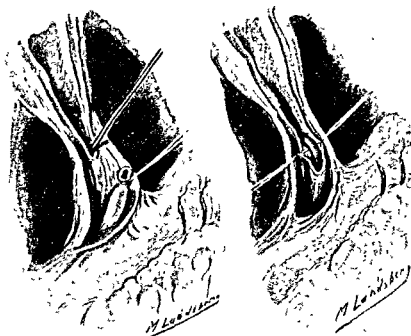


FIG. 126. Common duct opening directly into the gall-bladder. Only discovered after cholecystectomy. Repair as shown, patient well two years later (Pribram).

direct suture of the injured duct. But we must admit that sometimes this task is so difficult and even impossible that we are forced to secure the outflow by other means (implantation of the duct into the intestines, side-tracking operations).

TECHNIC OF THE SUTURE. It is most important that the anatomy be perfectly clear so that both stumps may be easily brought together. The tendency of the common duct to heal is favorable. Simple sutures or mattress sutures everting the inner coat can be used, so that the inner layers of the common duct come together.

Personally, I am in favor of fine silk as is used for sutures of arteries. It is kept ready with an eyeless needle in sterilized paraffin oil. Catgut sutures are usually too thick and enlarge the holes after operation during

the reabsorption to such an extent that leakage is more likely than when we use silk.

SUTURE OVER A RUBBER TUBE. In many cases, especially when the duct is narrow, the end-to-end suture can be facilitated when performing it over a rubber tube. One end of the rubber tube is introduced into the proximal end and the other into the distal end (Fig. 126). When the sutures are placed the tube can be removed before the threads are tied, but I found it always better to leave the rubber tube and to complete the whole suture over a so-called lost tube.

In these cases the distal end of the rubber tube should pass through the papilla into the duodenum to an extent of two or three inches. We can then be certain that the rubber tube will be expelled after a few days with the stool. It is advisable to observe the following rules:

(1) The rubber tube should not be too thick so that it can be easily moved.

(2) It should be thoroughly lubricated with paraffin oil in order to facilitate the expulsion. There is a definite advantage in leaving the drain. The suture is much safer through the absence of any pressure in the bile ducts. Leakage and stenosis in the part of the suture are very rare.

On the other hand, when no rubber tube is left and when there is leakage and the suture gives way in some parts, there is some danger of a shrinking through the formation of a bigger scar around the suture.

Over the first layer of the suture a second one should always be made using the surrounding tissue of the hepatoduodenal ligament, some fatty serous tissue from the neck of the gallbladder or even a free graft strip of omentum. In this way, even in the case of gaps and when the wall is friable because of inflammation, the suture can be made quite safe. I have had very satisfactory results with these sutures over a tube. Since I have used the technic of allowing the tube to reach far enough down into the duodenum, all these tubes have been discharged after a few days. In previous cases when this technic of having the distal end of the rubber tube to pass through the papilla into the duodenum was not yet observed, the rubber tube was still there after several years. I have followed up some cases in which such a drain has been observed at x-ray examination for months and even years without causing any trouble. I might also have chosen too thick a drain when operating. In one case—it was a colleague I operated upon—I have observed the drain still lying in its place five and a half years after the operation. He did not complain of any trouble. The following is an account of this case:

A stone, the size of a small hazelnut, had just passed through the enlarged cystic duct into the common duct, causing an extended necrosis in the wall of the common duct which was friable, so that when working

up the stone the wall was entirely torn. There was a big gap in the anterior and lateral wall and because of the inflammatory state of the surrounding tissue it was difficult to approach the two ends. A rubber tube was placed into the duct and I performed a suture as well as I could, but it was satisfactory only to a certain degree on the posterior and medial sides. The rubber tube was still to be seen through the gap in the sutured duct. With surrounding tissue and a piece of free graft omentum I tried to cover the gap. The distal end of the tube did not pass through the papilla into the duodenum. Another drain was left in the peritoneal cavity and the abdominal wall closed. Very little leakage of bile occurred in the next days. Recovery was uneventful. After five and a half years the x-ray examination showed the rubber tube still lying in its place without causing any trouble.

But on the other hand, I should like to mention another case in which a secondary operation may become necessary. The patient, a woman, died from pneumonia one year after operation. At the postmortem examination the rubber drain was found entirely incrustated. On the upper end there was found a stone with a funnel one-sixth inch wide, so that the bile could pass into the duodenum without any stasis. The patient had no trouble at all with her biliary system. However, it is certainly advisable that when operating over a rubber tube we should observe the technical rules which secure a discharge of the prosthesis after a few days.

I have injured the hepatic duct in the case of an anatomical abnormality in which a short hepatic duct emptied directly into the gallbladder (Fig. 124) and in which there was only one duct leading from the gallbladder into the duodenum. When the gallbladder was removed this single duct was entirely divided and had to be reconstructed by direct suture (Fig. 126). The following is the case report:

The patient, twenty years of age, had been operated upon on December 13, 1927, for an acute condition of the gallbladder. The abdomen was opened by a costal incision and the gallbladder was found deformed and in a state of acute inflammation. The gallbladder consisted of three parts separated from each other by transversal folds, so that a communication between these three parts had the width of a small finger. There was some turbid effusion in the abdominal cavity. The whole inside of the gallbladder was electrocoagulated and the neck of the bladder containing the proximal part of the cystic duct was removed. It seemed that the main hepatic duct was emptying into the gallbladder and the divided duct, supposed to be the cystic duct, appeared to be the common duct. The fact was that the cystic duct was entirely absent and a gap appeared between the hepatic duct the width of the whole gallbladder. The hepatic stump was only a quarter inch long. Repair was carried out in the following way:

A small rubber tube was introduced with its one end through the hepatic duct into the liver and the other end into the common duct, the ligature of which had been removed. Over this rubber tube which was allowed to pass down into the duodenum an end-to-end suture with fine paraffin silk was performed. The suture was covered safely with some serous tissue remaining from the gallbladder. The serous layers, remaining after the coagulation of all other layers of the wall of the gallbladder, were folded in and sutured together over the liver bed. The suture of the hepatic duct appeared to be so safe that the abdominal wall was closed without further drainage. Recovery was uneventful except for a slight jaundice which disappeared after a few days. The rubber tube was discharged with the stool after several weeks.

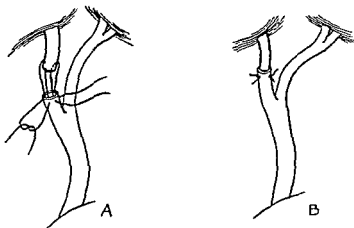


Fig. 127. Telescopic method of repair of the hepatic duct.

For similar cases, provided a longer hepatic stump was left, C. H. Mayo has advocated his telescopic method which was reported by Walters in 1924. Two sutures are placed in opposite sites of the proximal end of the cut hepatic duct. Both ends of the suture are passed into the lumen of the cystic duct, so that the hepatic duct can be telescoped into the cystic duct. Each suture is passed out through the wall so that the ducts can be held in position when the ends of each double suture have been tied together (Fig. 127).

There are cases in which the direct suture and reconstruction is either too difficult or not safe enough. This is the case when the injury is either too low down in the supraduodenal-retroduodenal part of the duct or too high up in the hepatic duct near the liver.

THE COMMON DUCT TORN IN THE SUPRADUODENAL PART: TECHNIC. An end-to-end suture of the common bile duct sutured over a rubber tube which is inserted through the papilla into the duodenum might still be performed fairly low down; this should be tried first.

When this procedure proves impossible or too difficult, an implantation of the proximal part into a new opening of the duodenum at a suitable place can be performed. But it seems better to ligate both ends of the torn duct and to perform a wide lateral anastomosis (choleodocho-duodenostomy). If the gallbladder has not been removed, the outflow of bile can also be secured by performing a cholecystoduodenostomy or a cholecystogastrostomy, provided the cystic duct is not damaged.

PLASTIC OPERATIONS FOR RECONSTRUCTION. These plastic operations are rarely necessary in the case of a fresh injury in which a direct reconstruction of the injured common duct is practically always possible. We have to perform such a plastic reconstruction mainly at a so-called secondary operation, that is to say, when the common duct has been injured at the first operation and the injury has been overlooked and the patient has to be operated upon again for the sequelae of this injury.

In the majority of cases the operation is unduly postponed, but one can understand that a surgeon is rather reluctant to advise a second operation soon after the first. On the other hand the cause of the complications mentioned above is not always quite clear. So there is still some hope left in the back of the surgeon's mind for spontaneous healing. The serious decision for a second operation is taken only after a long time when the general state of the patient definitely deteriorates. Then the necessity for an urgent operation arises and the patients are poor risks.

OPERATIVE PROCEDURES. Many surgeons advocate the use of a fresh incision and avoidance of the old scar so that an approach to the difficult area can be done through the free peritoneal cavity with the anatomy clearly in view. This seems to me unnecessary. First of all, I do not think that the new approach reveals the anatomy of the injured duct. On the other hand there is a definite advantage in limiting the operative field. (See, Local Immunity). I found it best to make an incision which leads directly to the operative field.

The exposure is more important than anything else. I always make the costal incision for these secondary operations no matter where the first incision might have been. Even in the presence of large adhesions and scars the clear exposure of the operative field is not too difficult.

The first task is to find the edge of the liver. It is sometimes adherent to the peritoneum, so we should always open the peritoneum first in a free part where there are not too many adhesions.

With the left forefinger as a guide we proceed safely toward the edge of the liver and carefully divide all adhesions. With the same forefinger we can easily distinguish between a scar and an adherent organ. In this way it is not too difficult to lay bare the whole undersurface of the liver. We separate the adherent pylorus, the duodenum and colon. When these organs are separated they are walled off with large abdominal pads

saturated in hot saline solution. A self-retaining retractor is inserted.

The difficult task is not so much the exposure of the operative field but the identification of the common duct. In all cases the big portal vein is a helpful guide to find the place of the common duct. When the duct has been injured very low down in its supra- or retroduodenal part and an obliteration or stricture has developed, the proximal end of the common duct can be found enlarged to the width of a thumb. In these cases not only the exposure but also the repair (anastomosis with the duodenum) can easily be performed. But in cases in which the middle or upper part of the hepatic duct has been injured, the identification of the obliterated distal end of the common duct can be difficult. This part downward from the scar can be obliterated and transformed into a fibrous band so that no duct and no lumen exists any more.

Furthermore, the shrinking of the scars may change the normal relation to other structures. When the duct is obliterated, the portal vein may easily be taken for the bile duct. An important rule is not to rely only on the structure when opening the assumed duct but to aspirate first the content with a fine needle.

The most difficult task arises when the whole duct from the duodenum up to the liver is transformed into a scarified fibrous band with only a small or no lumen inside. The hepatic stump left in these cases may be only one-half to one inch long. In trying to find the lumen we make longitudinal incisions through the fibrous tissue until we reach a greenish stained lumen. We try to introduce a probe downward and upward. But it is always easier to start with the exposure of the hepatic duct which, owing to enlargement, can be found easier. The aspiration of bile is always advisable before opening.

In some cases this part is transformed into a firm mass of scars. Then needling and aspiration are the only means to guide us to at least one of the hepatic ducts. Along this needle a small incision is made and a probe introduced downward. We should do our best to expose the hepatic duct as much as possible downward; every quarter of an inch we gain is of the greatest importance for the plastic operation we have to perform.

In these secondary operations the distal part of the duct is rarely fit for a direct suture, as in cases immediately after an injury in which these direct sutures can be applied. If plastic operation has to be performed, the kind of operation depends on special conditions and consists in the implantation of the hepatic duct either into the stomach, the duodenum or into another intestinal loop. The gallbladder has in most cases been removed in the first operation, so it can not be used any more for the purpose of securing bile drainage into the intestine. When a certain length of the hepatic stump has been saved, the direct implantation over a rubber or vitallium tube is the best procedure.

The rubber tube is introduced first into the hepatic duct. A small incision is made on the stomach or duodenum, the lower end of the rubber tube is introduced and a direct suture between the hepatic duct and the duodenum or stomach is performed. The rubber tube which can be very small in these cases can then be pushed down toward the stomach or the duodenum.

The suture is reinforced by a second row, suturing over it surrounding tissue or omentum. When we do not feel quite safe about the suture, it is wise not to push down the rubber tube over which the suture has been performed but to leave it for a spontaneous discharge which usually takes place after a few days.

Drainage of the abdominal cavity is not necessary but it must be left to the judgment of the surgeon as to how he feels about the control of bleeding from the scars and the safety of the suture. No harm is done when a drain is left as long as it does not come too near to the suture. In case a very short hepatic stump is left Voelcker's method has proved very satisfactory.

VOELCKER'S METHOD (Fig. 128). A rubber tube is introduced into the hepatic stump and firmly fixed with a catgut suture. A small incision is then made into the duodenum, a forceps introduced and pushed upward. Over this forceps another small incision is made and the lower end of the rubber tube is grasped and pulled through both incisions through the duodenum. The duodenum is pushed upward over this tube so that the hepatic stump comes down into the lumen of the duodenum. The outer wall of the duodenum is sutured around the hepatic duct with interrupted sutures or by two purse-string sutures. The rubber tube has a sidehole inside the duodenum so that the bile drains into the intestines. The lower hole in the duodenum is either directly closed with purse-string sutures around the tube or, as Voelcker has advocated, in the form of a Witzel canal. The lower end of the rubber tube is allowed to pass through the incision of the abdominal wall. After a certain time the rubber tube can easily be removed and the fistula will close.

I had good success with this technic in a very difficult case. The implantation of the short hepatic stump was made into the stomach (Fig. 129).

In the first operation the hepatic duct was apparently divided high up under the liver. The patient was admitted for a reoperation with jaundice lasting for more than five months. The jaundice had set in shortly after an operation for gallstones. It was supposed that an overlooked stone had led to another obstruction. The operation was postponed in the hope that the stone would pass spontaneously the papilla. When operating, it was difficult to recognize the normal anatomy. In firm scars of fibrous tissue no common duct was found at all, only a fibrous band replacing the former common duct. It was a total obliteration.

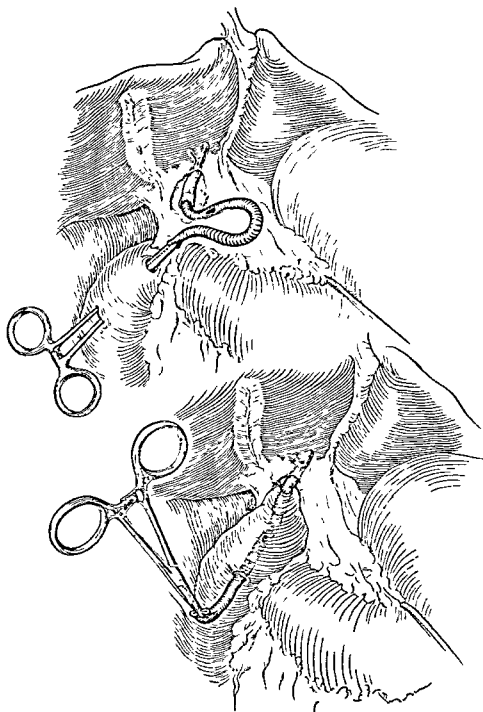


FIG 128 Hepatoduodenostomy—Voelcker method.



FIG. 129. Implantation of the hepatic duct into the stomach over a rubber tube.

tion. A stump of the hepatic duct, one-half inch long, was at last found. It was necessary somehow to suture this stump into the stomach, but any attempt at direct suture resulted only in damaging the wall of the hepatic stump and in shortening it more and more in this way. A small rubber tube was then introduced through the hepatic stump. A catgut suture was laid right through the wall of the hepatic stump and the ends of the suture tied around the rubber tube. In this way the approximated stump was firmly fixed on the rubber drain and I had a good grip to pull down the hepatic stump into a button hole over the stomach. This was done in the following way: One button hole incision was made in the stomach for the implantation of the duct. The rubber tube was allowed to pass through another hole near the greater curvature. A purse-string suture was laid loose around the upper hole in the stomach. Three fine catgut sutures were laid through the hepatic stump, rubber tube and wall of the stomach and in this way the stump was fixed. Over these sutures and the stump the purse-string suture was tied, so that the stomach came in close contact with the undersurface of the liver. The lower end of the rubber tube, which was held by the assistant during the whole operation, was cut and allowed to slip into the stomach. The opening in the stomach was then closed. A small rubber drain for drainage of the abdominal cavity was introduced for safety near the pylorus. Recovery was uneventful; no leakage of bile took place. The rubber tube was discharged three weeks after operation. The new bile duct showed no symptoms of biliary obstruction in the follow-up.

Another satisfactory plastic method of implantation of a very short common duct into the duodenum has been described by G. Walton:

WALTON'S METHOD (Fig. 130 upper half). Walton reconstructed the common duct by the use of a pedunculated flap formed from the duodenal wall. In this case the common bile duct was found obliterated after an operation for common duct stones. A tongue-shaped flap pedunculated in its lower end was cut from the duodenal wall and reflected downward. The gap was closed except for an opening about the size of the bile duct at the basis of the flap. A rubber tube was introduced into the stump of the bile duct with its lower end in the opening of the duodenum. The flap was reflected upward and sutured to the anterior part of the bile duct, while the lateral margins of the flap were sutured to the duodenum along the side of the rubber tube. In this way the artificial lower end of the bile duct was lined on its posterior aspect with a serous surface of the duodenum, while the anterior wall was formed by the mucous membrane of the flap.

Walton's method was used and recommended also by W. J. Mayo. Moynihan has modified this method (Fig. 130, lower half). He passed the flap which he had pedicled on its upper end completely round the tube so that this artificial common duct was lined throughout with mucous

membrane. He has applied this method in three cases. Transplanted grafts with fasciae, or by segment of a vein (jugular vein) or by a calf's artery have not proved useful in experimental work.

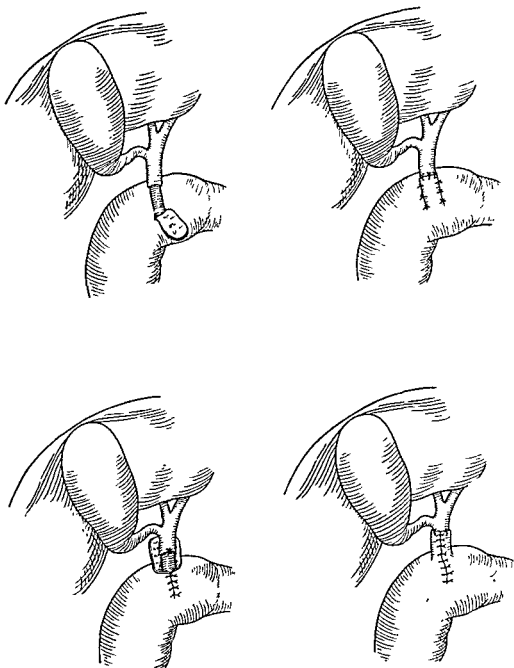


FIG. 130 Plastic reconstruction of the common bile duct. Upper half. Walton method.
Lower half. Moynihan method.

In cases in which there is practically no stump of the hepatic duct or only an opening in the lower surface of the liver the following plastic method developed by Goetze has proved successful:

GOETZE'S METHOD (Fig. 131). From the anterior wall of the duodenum a rectangular flap pointing to the liver, one inch long with a basis of one-half to one inch, is formed. The opening into the duodenum is closed leaving only a hole the size of the hepatic duct. Meanwhile the

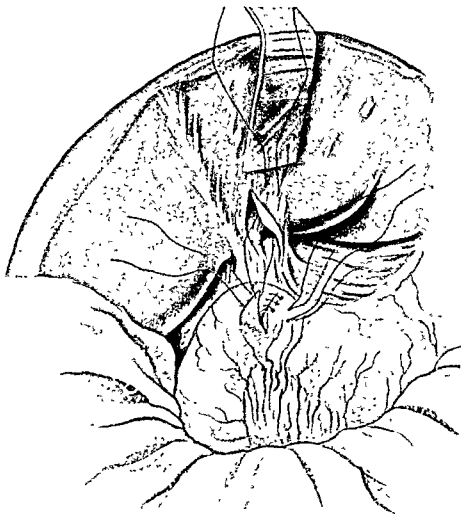


FIG. 131. Direct anastomosis between the liver and duodenum Goetze's method

hepatic duct is dilated and split upward into the liver, so that a hole results into which the flap can be inserted. This is done in the following way:

A thread through the point of the flap is armed with two needles passed into the split hepatic duct and knotted on the anterior surface

of the liver. In this way the duodenal flap is pulled into the hepatic duct, the serous part being close to the divided liver tissue over the hepatic duct. The duodenum is fixed to the undersurface of the liver. This operation can be done with or without a rubber tube which should not be longer than one to two inches for these cases.

This method has been used in six cases with two deaths. The follow-up of the patients showed satisfactory results with no cholangitis.

INTRAHEPATIC INJURY OF A HEPATIC DUCT. This injury may be caused by a deep rupture but in most cases is caused by a gunshot wound as in war injuries. In these cases the injured hepatic duct is found in the depth of a large hole. A bile fistula often results in cases otherwise healing.

TREATMENT. A proper suture of the intrahepatic duct can rarely be made. I have successfully treated such a case by performing laparotomy opening the common duct and introducing a rubber drain into the upper end of the injured hepatic duct and farther into the liver. The common duct was sewn watertight around the rubber tube; the other end of the drain was brought outside the abdominal cavity.

The liver wound was packed with gauze soaked in olive oil and the greater portion of bile drained outside. The leakage from the liver wound diminished at once and the wound was closed after three weeks. Then the drainage tube was removed and the patient was dismissed with both fistulae closed.

In another case I introduced a small rubber drain having a number of side holes. One end was passed into the central part of the hepatic duct and the lower end was passed through the papilla into the duodenum. The rubber drain was fixed with a catgut suture, the ends of which were left long and fixed with adhesive plaster to the skin so that the rubber drain would not be discharged too early. The liver wound healed and the whole length of the rubber drain was discharged with the stool after three weeks.

CHOLANGIOENTEROSTOMY (KEHR-ENDERLEN). When no hepatic stump is found at all a direct communication between liver and duodenum or jejunum can be performed. An incision is made on the undersurface of the liver not too near to the hylus in order to avoid larger blood vessels. This incision is done by diathermy. When a large bile duct has been found, a small rubber tube with a side hole is introduced, one end of which is allowed to pass into the jejunal loop or better into the stomach. The stomach is carefully sutured around this liver opening.

But it must be said that the results of this operation are not very satisfactory. If dissection is carried out carefully the main hepatic duct can practically always be found in the scars, so that an anastomosis of this hepatic duct with the duodenum or the stomach by one of the previously described methods can be performed.

POSTOPERATIVE BILE FISTULAE. Bile fistulae when lasting for a longer time may also become a matter of urgent surgery. Fistulae after cholecystostomy are not discussed here; they do not require urgent surgery except when combined with a total obstruction of the common duct after an injury of the bile duct. Bile fistulae as a result of stones will be discussed in a special chapter.

The deteriorating general state of the patients, progressive anemia, general atrophy, especially osteoporosis, make the operation urgent and must always be performed even in poor risk patients.

Cholangiography has proved very helpful in producing evidence for the cause and the position of the obstruction. It should always be done before operating. A small rubber tube is introduced as far down as possible into the fistula and lipiodol injected on the x-ray table while the photograph is being taken. The reasons for the obstruction can be given as follows: (1) a stone which had been overlooked; (2) a cicatricial stenosis; (3) an operative injury to the bile duct; and (4) a malignant growth.

In many cases the cicatricial stenosis was due to the application of drainage with a T-tube. When removing this T-tube weeks later, the bile duct already in a state of cicatrization was torn again. This secondary injury results very often in a stricture and bile fistula. But these cases become rather rare, since most surgeons have abandoned the T-tube.

TREATMENT. The treatment depends on the position of the fistula and the size and length of the common duct. One or another of the above mentioned plastic operations can be performed in suitable cases. As a special operation for such bile fistulae, a procedure must be discussed here in which a track of the fistula itself is used for an implantation into the intestine, into the stomach or into another intestinal loop.

A probe is introduced into the fistula and the external opening is circumcised and grasped together with the probe by forceps. Then the whole track is mobilized. It is advisable to mobilize this track not more than necessary because the blood supply is sometimes poor. It should be mobilized so that the implantation into the intestines can be performed easily, the track being twisted and bent without any tension. The external skin portion of the fistula can be excised or also left.

INJURIES TO THE HEPATIC VESSELS: INJURIES TO THE HEPATIC ARTERY. Injuries to the hepatic artery by external force, stab wounds, gunshot wounds, etc., have a high mortality even when immediate operation follows the injury. The bleeding and the loss of blood is always severe. On the other hand when the bleeding is controlled by ligation of the hepatic artery the danger of necrosis of the liver is considerable. But this fact depends upon the place where the artery has been injured

and whether a so-called accessory hepatic artery is present. This occurs in about 25 per cent of the cases.

There are instances reported in which the ligation of the hepatic artery has been performed without any damage. Ligation of the common hepatic artery proximal to the division into the right and the left hepatic arteries and proximal to the gastroduodenal artery can be performed without any damage to the liver, because the great amount of anastomosis is sufficient to secure the necessary blood supply.

Injury to the cystic and hepatic arteries is not infrequent when operating upon the cystic duct during a cholecystectomy. The following are the dangers to be avoided:

(1) When the cystic artery is ligated and divided between two ligatures, the ligature slips sometimes from the proximal stump while manipulation is carried on in this region. It is pushed away especially by the retractor with which the assistant holds the liver. The bleeding may be serious. The surgeon tries to grasp the vessel without having a good view of the field because the operative area is constantly filling with blood. Injury can be done in these cases to the hepatic artery as well as to the portal vein and to the hepatic duct. In such a mishap it is much better to check the blood supply by compressing the hepatoduodenal ligament with two fingers of the left hand and to use an artery clamp when the cystic artery is clearly seen.

But I think the best way to avoid such a mishap is not to divide the cystic artery at all. A simple ligature of the cystic artery is sufficient, for the serous layers in the neck of the bladder are preserved. A subserous excision may be done, or in difficult cases an electrocoagulation.

(2) In cases of abnormalities the cystic artery or hepatic artery can be divided in an unusual place. The laying bare of the anatomy is the best safeguard against such a mishap which may have serious consequences.

(3) **TRAUMATIC ANEURYSM OF THE HEPATIC ARTERY** is a third danger. When suturing the common duct or through the serous tissue of the gallbladder, the point of the needle may injure the wall of the hepatic artery. Bleeding may not take place at once but damage to the wall is sufficient for the development of an aneurysm. The following case seems to be worth mentioning:

A young woman, twenty years of age, was operated upon for acute cholecystitis (strawberry gallbladder), with a subserous excision and primary closure of the abdominal wall on September 19, 1932. The patient did not feel well the whole time after operation and was brought to the hospital after a sudden severe hematemesis which occurred when she was walking on the street. A blood transfusion was given and the patient recovered. A gastric ulcer was assumed as the cause. But an

x-ray examination did not show any sign of this and in spite of diet there was always occult blood in the stool. On January 7, 1933, (three months later) severe hematemesis occurred again. A blood transfusion of 350 cc. was given with good results. On January 24, another hematemesis occurred and operation was decided upon. This was performed on February 13. A laparotomy incision was made in the median line, the duodenum was displaced, and a tumor the size of a chicken's egg was found behind the choledochus. Isolation of the tumor revealed a pulsating aneurysm belonging to the hepatic artery. There was a communication between the aneurysm sac and the common duct. Ligature of the artery and several vessels was done and the patient had an uneventful recovery.

It must be assumed that when operating the first time the hepatic artery was injured by a needle, an aneurysm developed and recurrent hemorrhages occurred through the common duct.

There are two other cases of traumatic aneurysm reported in recent literature (Ecarius and Küntschner).

INJURIES TO THE PORTAL VEIN. Injuries to the portal vein through external force are observed through stab or gunshot wounds. The mortality is very high. A ligature to control hemorrhage is definitely fatal and compression by plugging gauze is also no method which promises success. Thrombosis of the portal vein is the inevitable sequel and the result is eventually the same as when a ligation is performed. The only way to save the patient is to make a proper suture of the opening. From a technical point of view these sutures are not too difficult.

Surgical injuries to the portal vein had been observed on several occasions, especially in dense scars during a secondary operation.

As already mentioned, scars can either displace the common duct which then overlies the portal vein, or, when there are only scars left of the common duct, the portal vein may be mistaken for the common duct and be opened. The best safeguard against such an injury is aspiration through a fine needle when any doubt arises before the opening of the assumed duct.

In order to perform a proper suture of the portal vein partial compression with the assistant's fingers or by use of a clamp is indispensable. The curved clamp as used in Trendelenburg's operation for pulmonary embolism can be applied.

In case one of the branches of the portal vein at the porta hepatis is injured its ligation can be performed. Infarction in the respective liver lobe or in a part of it may happen, but the patient will likely recover.

There are cases reported (Nauwark) in which, after occlusion of the right branch of the portal vein, the right liver lobe has shrunk to the size of a fist while the left lobe hypertrophied to the size of a normal liver.

LIVER ABSCESES

GENERAL SIGNS. The diagnosis of liver abscesses is not easy. Stormy and striking clinical signs are not regular; the development may be rather slow. Nevertheless early diagnosis and early operation can be life-saving. I would not say that liver abscesses, especially solitary abscesses, cannot heal without surgical interference. We undoubtedly see sometimes the remnants of a healed liver abscess or at least an abscess which has become avirulent without any signs of spreading and without causing further necrosis.

But more often we find at the postmortem examination liver abscesses which were the cause of the death of the patient who has died of septicemia without a detection of its origin and in which the surgical opening of the liver abscess could have saved the patient's life.

Gradual enlargement of the liver in the course of septicemia strongly suggests the development of a liver abscess. Pain is especially marked in cases of superficial location, otherwise only a general tenderness over the liver is observed.

While breathing, pain is felt when the abscess lies under the convexity, near the diaphragm. Pain can radiate to the right shoulder; the phrenic symptom is also frequently present. Tenderness in the intercostal spaces between the eighth and tenth ribs in the axillary line is very common. Characteristic of the enlargement of the liver due to a liver abscess is its development upward, thus raising the diaphragm. This upward enlargement can be demonstrated by x-ray examination. In its later stage the enlargement of the liver is also felt below the costal arch.

High leucocytosis is practically always found in the early stages. When an abscess becomes incapsulated, the blood count may be normal again. The following are the main causes for the development of a liver abscess: (1) after injuries (gunshot wounds); (2) through arterial dissemination; (3) abscess following cholangitis; (4) infection through the portal vein; (5) tropical abscesses; and (6) retrograde infection.

Liver abscesses and their treatment following gunshot wounds are discussed in the chapter on war injuries.

Liver abscesses spread through the arterial course are rare. The main cause is ulcerous endocarditis. But there are cases quoted in the literature in which a liver abscess has developed in the course and sequel of erysipelas, prostatic abscesses and other pyemic conditions. These cases are practically always fatal and rarely offer an occasion for surgical intervention.

Under abscesses originating from the biliary tract we may distinguish (a) abscesses following a suppurative cholecystitis. These abscesses are found in the liver bed or in the surroundings of the gallbladder as sequels to localized rupture into the liver. They are seated sometimes

deep in the liver containing stones and are frequently overlooked when a simple cholecystostomy is performed as an operation of emergency in the case of a ruptured bladder. This is one of the reasons why I have abandoned cholecystostomy in all these cases since the radical electro-surgical operation has proved very successful also in dealing with these liver abscesses. Indeed it should be adopted as a routine method for such cases.

(b) The development of abscesses originating around the intra-hepatic bile ducts is practically always due to a combined cause: cholangitis plus obstruction of the common duct. While an infiltration with leucocytes is a very common finding in most cases around the small bile ducts, an acute stone obstruction favors sometimes quick development into a real pericholangitic abscess.

Pathology and treatment of these cases are fully described in the chapter on acute obstruction of bile ducts. While a stone is in most cases the cause of an acute obstruction, the following case of liver abscess may be mentioned in which a liver abscess followed an obstruction caused by an ascaris invading the common duct:

A man, thirty-four years of age, was admitted for urgent surgery. He was jaundiced and his temperature was 101.5° F. Below the xyphoid process a very tender swelling following respiratory movement was felt. Under the diagnosis of liver abscess the patient was operated upon. The abdominal wall was incised over the swelling and an abscess opened in which there was badly smelling bile-stained pus. Two days after the operation an ascaris was found in the cavity. The general state of the patient deteriorated. Jaundice increased and the temperature remained high. Colicky pain set in and the stool became acholic. It seemed probable that an occlusion of the common duct through the ascaris had taken place. A week after the first operation the second operation was performed. The gallbladder was found to be enlarged as well as the common duct. This duct was incised and an ascaris, 25 cm. long, filling the whole common duct with its tail down to the papilla, was removed. Another ascaris of a smaller size was found in the hepatic duct. A communication of the common duct with the liver abscess could not be found. A sub-serous excision of the bladder, which contained a few stones, was carried out and the common duct drained.

In the days following the operation the patient vomited ascarides twice. Santonin was administered and the drain in the common duct was removed on the tenth day. Following this, recovery was uneventful.

The portal vein is the main entrance for metastatic abscesses. They may originate in suppurative processes from which the portal vein carries the venous blood. They may develop with or without a pyelephlebitis. In the latter case they originate from an infected embolus passing

through the portal vein and penetrating directly into the liver where they stop intrahepatically in one of the small branches of the vein. In other cases the infection spreads first to the vein causing a pylephlebitis.

Suppurative Pylephlebitis

ETIOLOGY. Infection of the portal vein with suppuration and thrombosis is frequently due to a primary suppurative appendicitis, particularly when an appendicular abscess is under pressure. According to Weir and Bever, 19 per cent of twenty-eight cases were due to appendicitis and 28 per cent followed inflammation of the biliary tract.

On the other hand, statistical figures on the frequency of a suppurative pylephlebitis have shown that with the increased tendency toward early operation the frequency of pylephlebitis as a complication of appendicitis has dropped. Leaving aside appendicitis and infection of the biliary tract, ulcerative colitis, diverticulitis, malignant tumors of the rectum and suppurating piles can be mentioned as rare causes.

PATHOLOGICAL FINDINGS. The portal vein is found enlarged and filled with suppurating adherent thrombi, while the wall is involved in the inflammation as well as the surrounding tissues. Suppurating mesenteric glands are frequent. In the majority of cases the infection has already spread to the liver in the form of multiple metastatic abscesses.

CLINICAL SIGNS. In the course of appendicitis, either in the beginning or a few days later or even after the first symptoms have already subsided, sudden chills set in followed by high temperature. High leucocytosis is found in the blood; the liver becomes tender and enlarges gradually. The patients give the impression of being very ill and in a grave septic state. Slight jaundice is frequent as is also diarrhea. The fatal outcome occurs in the majority of cases partly due to general septicemia or partly due to general suppurative peritonitis following the rupture of a suppurative mesenteric gland or the leakage of a superficial liver abscess into the peritoneal cavity.

TREATMENT. Surgical intervention offers little hope for recovery. Only in those rare cases in which a solitary abscess in the liver is formed, which can be opened (Petren), or when a subphrenic abscess develops, which can be drained, the patient may recover. The greatest hope for recovery of patients with pylephlebitis is administration of penicillin and sulfa drugs. This should be tried for two or three days, giving penicillin up to 300,000 units daily and of sulfadiazine up to 60 grains daily. As far as surgery is concerned, the best method of treatment, of course, would be a preventive measure consisting in the ligation of the mesenteric vein before a real pylephlebitis develops.

The indications for this operation are, however, very difficult. There is hope of success with this measure only when the mesenteric vein is

ligated after the first suspicion of a suppurative thrombosis following appendicitis. This is certainly the case when a sudden chill in the course of such a disease sets in.

In all cases before operating upon the patient he should be examined thoroughly for the presence of a subphrenic abscess, clinically and by means of x-rays. There is no purpose in operating when an enlargement of the liver indicates that a liver abscess has already developed.

TECHNIC. In most cases the appendix has already been removed with or without drainage. This area of the first operation should be protected thoroughly with gauze and oil silk and a laparotomy performed with a midline incision below the umbilicus or with the medial pararectus incision.

The mesenteric vein concerned should be ligated as closely as possible to the junction with a superior mesenteric vein. In this way the danger of gangrene following congestion can be best avoided. As these cases must be considered practically hopeless, a ligature of the superior mesenteric vein higher up can be attempted. However, even if surgery is resorted to, it should be supplemented by administration of penicillin and sulfa drugs.

Tropical Abscesses

A tropical abscess is said to be found in 3 to 10 per cent of patients with amebic dysentery. It is much more common in Europe than in the United States, and is found particularly in men. Only 10 per cent are found to be in the left lobe against 90 per cent in the right lobe. In the majority of cases the abscesses are found in the upper and posterior part. They are never multiple. Infection takes place via the inferior mesenteric and portal veins. The ameba reaching the liver produces necrosis with extensive liquefaction of the liver tissue. Abscesses containing up to 7 to 8 liters of pink or anchovy paste-like fluid have been observed. Practically no secondary infection takes place.

Adhesions between the liver surface and pleura followed by perforation into the pleural cavity occurs in 25 per cent. Abscesses may rupture into the bronchi and heal in this way. Rupture into the peritoneal cavity is rare.

There is practically no chance of spontaneous healing through reabsorption. Suspicion of the presence of an amebic abscess arises, especially in tropical countries, in all cases in which there is an undefined enlargement of the liver.

TREATMENT. Urgent surgical intervention may be necessary in cases with a rapidly enlarging abscess. In case of rupture the abscess burst into the peritoneal cavity, into the sub-diaphragmatic spaces or into the pleural cavity through the diaphragm.

There are cases reported in which a large amount of the anchovy sauce-like pus has been expectorated in an agonizing attack of cough

after which the abscess healed. In other cases pyopneumothorax results.

It must be stated that in all cases of rupture the mortality rate is very high. This fact suggests the necessity of early treatment.

The special diagnosis is made only when amebic dysentery has been diagnosed previously. The treatment consists in emptying the abscess and administering emetine. Since the introduction of emetine the mortality rate has dropped from about 56.8 per cent (Rogers, 1913) to 14.4 per cent (Rogers, 1922).

As for the surgical procedure, two methods are applied: Opening and drainage of the abscess or emptying the abscess by aspiration.

In all cases the cavity should be washed out with emetine solution. Most surgeons today with experience in the treatment of tropical abscesses favor the method of aspiration and irrigation with emetine.

When the abscess is opened by incision, a suture after the treatment is preferable to open drainage. When emptying the abscess, it is advisable to avoid too rapid an emptying. This is a well founded rule in pleural empyema, in ascites and in all cases in which the collection of a large amount of fluid has to be removed.

As for ways of approach, the abscess is always incised or aspirated over the most prominent part of the swelling. In case the abscess is located higher up in the convexity, the ways of approach are similar to those which will be described under subphrenic abscesses.

Without having tried the electrocoagulating method in tropical abscesses, I would imagine that in these cases also electrocoagulation may prove advantageous.

The emetine treatment consists of the intramuscular injection of emetine hydrochloride (one-fifth of 1 grain daily). Emetine can also be administered orally in the form of emetine bismuth iodide.

In rare cases a middle ear infection (mastoiditis) with or without thrombophlebitis of the jugular vein may be followed by a liver abscess. The direct retrograde way of infection seems probable due to the fact that in these cases only liver abscesses have been found and no abscess in the lung or a general septicemia. These cases may be mentioned only as a curiosity and have no practical value for the surgeon.

Subphrenic Abscess

Though the incidence of subphrenic infection is high, not in all cases does a real abscess develop demanding urgent operation. The frequency in males is higher than in females at a ratio of 3 to 1. Disregarding local infection following an injury (gunshot wounds), perforated appendix or perforated ulcers of stomach and duodenum are the causes in 50 to 60 per cent of the cases.

The following scheme (Ochsner) presents a summary of the main causes of origin:

(1) Subphrenic abscess following an injury in the immediate vicinity.

(2) From distant portions of the peritoneal cavity right iliac fossa and pelvic infection may drain into the subphrenic area. From the right iliac fossa infection may extend through the gutter between the ascending colon and the lateral parietal peritoneum upward to the subphrenic space.

(3) Perinephritic abscess in the retroperitoneal cellular tissue may extend upward to the extraperitoneal subphrenic area.

(4) Spread by the retroperitoneal lymphatic vessels (retroperitoneal lymphangitis) (Munroe).

(5) Lymphangitis along the deep epigastric artery.

(6) Rupture of liver abscess (a) following portal thrombophlebitis; (b) cholangitic abscess; (c) amebic abscess.

(7) Perforation of gastric or duodenal ulcer.

[(8) Rupture of gallbladder.

[In some rare cases the infection calms down so that the subphrenic abscess is a mere collection of a more or less nonvirulent or even sterile liquid giving no alarming symptoms. There are cases reported (Russell) in which a subphrenic abscess was diagnosed only seven months after the perforation of a peptic ulcer; in another case, one year after pneumonia and in a certain case even seven years after an empyema. However, these cases of spontaneous healing are very rare and in most instances the development of a subphrenic abscess is followed by alarming symptoms and must be considered as a matter of urgent surgery.

The onset of an abscess may be very abrupt like in an acute intra-abdominal suppuration. About 35 per cent are of this type. In about 40.6 per cent the onset is insidious in the course of an obscure intra-abdominal lesion, and correct diagnosis often is missing. We should think of the possibility of such an abscess, then a thorough search will enable us to establish the right diagnosis.

CLINICAL SYMPTOMS. The patient is taken seriously ill with constant or fluctuating temperature, giving the impression of a grave infection. The history of a previous suppurative disease, intraperitoneally or retroperitoneally, (perinephritic abscess), strongly suggests the diagnosis, especially when, after emptying the abscess, the temperature starts to rise again a few days later.

Clinically we find elevation and immobility of the diaphragm. X-ray examination proves the existence of the subphrenic abscess in practically all cases. Fluid level with a collection of gas is very frequent. The roentgenogram or fluoroscopy is made in an upright position. Aspiration of pus with a long needle should be attempted only with the greatest care. Many cases of spreading infection following such attempts have been reported. But when there is a definite dullness over the phrenicocostal

angle, no harm can be done by careful aspiration over the dullness with a needle that is not too long.

In some cases a clear or only slightly turbid liquid can be aspirated from the pleural cavity as a collateral effusion. When pus is aspirated, there is a strict rule not to remove the needle, not to aspirate more than a few cc. and to operate immediately.

The following aphorism (Barnard) may give a useful diagnostic hint: Signs of pus somewhere, signs of pus nowhere else, signs of pus there

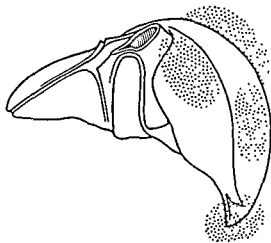


FIG. 132. Common locations for right subphrenic abscesses.

(i.e., in the subdiaphragmatic space.) This may include also a hidden abscess of the liver.

TREATMENT. Considering the poor state of the patient who becomes worse day by day, operation should be performed as early as possible, i.e., when the diagnosis can be made. However, before operating, a fair trial with penicillin and sulfa drugs should be given at least for two days. The following diagram shows the most frequent locations for subphrenic abscesses (Fig. 132). The right superior and posterior space behind the coronary ligament is predominant.

When opening the abscess the following rules should always be observed: (1) Approach by the shortest possible way; (2) make a wide opening so that a retraction of the cavity is facilitated; (3) provide for proper drainage from the lowest point; and (4) care should be taken to avoid contamination of uninvolved serous cavities, the pleura and the peritoneal cavity. This point must be considered as being of the utmost importance.

Ways of Approach

- (1) Transthoracic way:
 - (a) transpleural;
 - (b) extrapleural.

- (2) Transabdominal way:
 (a) transperitoneal;
 (b) extraperitoneal.

As above mentioned, the extraserous approach has always to be attempted and can be achieved in different ways. In quite a number of cases the extraserous approach is already prepared through inflammatory adhesions firmly separating the abscess from the serous cavity. The phrenicocostal angle can be entirely obliterated. The diaphragm has been lifted up and through the pressure of the abscess the diaphragm becomes firmly adherent to the pleura. So we can reach the abscess by the transthoracic and transdiaphragmatic way without contaminating the pleural cavity.

If adhesions do not exist, they can be artificially produced in the following ways: (1) in a one-stage operation by stitching together the diaphragm with the pleura in the whole extent where the opening of the abscess is intended; (2) in a two-stage operation. The first step of the operation is to produce adhesions either by padding with gauze or with a paraffin plumb.

TECHNIC OF TRANSTHORACIC AND TRANSDIAPHRAGMATIC APPROACH. The incision is made along the ninth rib. According to the special case, pieces of rib from the seventh to the tenth are resected without injuring the pleura. The intercostal muscles are ligated with the vessels at the places of resection of the ribs and cut away (Fig. 133). The costal pleura is now laid bare. We can satisfy ourselves now whether or not there are some adhesions between pleura and diaphragm when observing the respiratory movements.

If such adhesions are present the wound is walled off with gauze and we try to find a way to the abscess by aspirating with a firm needle. When we locate the abscess, an aspirator is introduced along the needle and the abscess is emptied.

We must always bear in mind that in most cases gas is collected over the abscess. The patient lies on the table on his left side or in a half upright position so that we may easily approach the gas-filled cavity. The characteristic smell of the aspirated gas indicates that we are in the right place.

The forefinger is now introduced and the diaphragm incised with the finger as a guide along the fibers to an adequate extent. With a rubber tube having the thickness of a thumb the cavity is drained.

In case of recent abscess where the wall of the cavity is not too firm, the external wound may be partially closed with a few deep interrupted stitches. Larger cavities should be packed with several pieces of gauze or by means of a Mikulicz tampon moistened with peruvian balsam.

A one-stage operation can also be performed, when no adhesions exist, in the following way:

(1) The pleura is stitched together with the diaphragm by continuous or interrupted suture. If the pleura should tear, it is a good procedure to use the excised pieces of the intercostal muscles to reinforce the stitches by sewing them over the torn pleura. The wound is thoroughly walled off with gauze and then the abscess is punctured by a needle, the pus aspirated by an electric aspirator and the diaphragm incised, packed with gauze and drained.

It must be emphasized that the danger of empyema following this

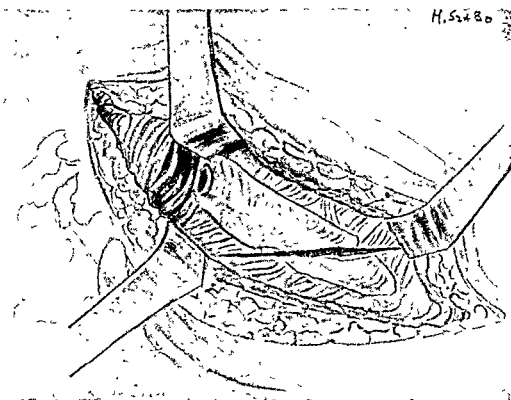


FIG. 133. Subphrenic abscess. Transthoracic, transdiaphragmatic approach.

one-stage operation is considerable. Ochsner has collected 305 cases drained transpleurally with a mortality rate of 39 per cent, sixteen of his own cases showing a mortality rate of 50 per cent.

TWO-STAGE OPERATION. First stage: (a) padding with gauze. The whole wound is firmly filled with gauze pushing the pleura toward the diaphragm. The wound is entirely closed over the gauze. (b) Paraffin Plumb Method. The same adhesions can be provoked in a first-stage by use of a paraffin plumb. It consists of:

Solid paraffin (melting point 50° C.)

75.0

Solid paraffin (melting point 43° C.)	25.0
Carbon bismuth	1.0
Vioform	0.05

In order to make the plumb easily visible in the x-ray picture a tablespoonful of sulphurated barium may be added. This paraffin is put into water of 55° F. so that a semisolid porridge is formed which can be moulded in the hand according to the shape of the cavity.

This method has proved very useful as a first-stage operation before opening an abscess not only in the subphrenic space but also in the lungs. The costal pleura is worked away gently with the finger from the costal

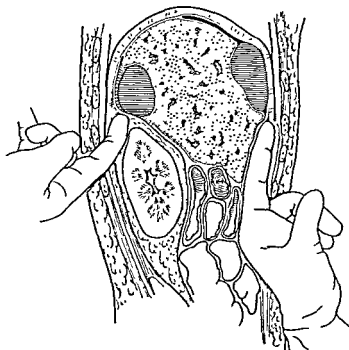


FIG. 134. Subphrenic abscess. Anterior and posterior approaches

frame around the wound. A large cavity is formed in this way; the paraffin plumb is inserted, moulded with the hand so that it fits in properly. The wound over the plumb is entirely closed. The costal pleura is in this way firmly pushed against the diaphragm. Firm adhesions are formed in a few days. Between the fifth and eighth days the second-stage operation is performed, the plumb removed and the abscess safely opened in the way previously described.

TRANSABDOMINAL APPROACH. Though the transthoracic way can be considered as a routine procedure in dealing with subphrenic abscess, there are cases in which a subdiaphragmatic approach may be considered

advantageous (Clairmont, Ochsner). These are cases in which an abscess lies more anteriorly spreading upward from the right renal pole.

In case of suspected subphrenic abscess which cannot be confirmed by x-ray film or exploratory puncture, transabdominal approach is preferable to the transthoracic one.

Because of the main locations of these abscesses—anterior or posterior to the coronary ligament—two ways of approach are available: the anterior and the posterior.

Under no circumstance should a transperitoneal drainage be per-

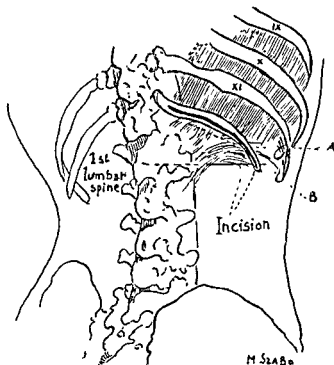


FIG. 135. Subdiaphragmatic abscess. Posterior approach.

formed. When operating and draining a subphrenic abscess by the subdiaphragmatic way, only the extraperitoneal drainage should be used.

a. **Anterior Approach** (Fig. 134). The skin incision, four inches long, is made below or, better, upon the costal arch (similar to the costal incision). External, internal and transverse abdominal muscles are divided, avoiding the opening of the peritoneum. When there are deeper situated abscesses, we frequently encounter tissue here becoming edematous. With a retractor the costal arch is lifted while the finger works gently upward separating the fascia transversalis and the peritoneum from the muscular portion of the diaphragm. In this way the upper surface of the liver is gradually approached and we soon locate the abscess lying between the upper surface of the liver and the dome of the diaphragm.

If in doubt, aspiration with a strong, curved needle is always helpful in pointing the way.

b. **Posterior Approach** (Fig. 135). When the abscess is suspected behind the coronary ligament, a posterior approach must be performed. The incision is made along the twelfth rib, which is resected subperiostally. Care should be taken not to injure the pleura. Muscles are divided transversely at the level of the first lumbar vertebra down to the renal fascia. With the help of the finger we work upward until the dome of the diaphragm is reached.

The advantages of this method are: (1) the avoiding of the opening of the pleura with its drawbacks of pneumothorax and infection; (2) the drainage is allowed to be carried out in the lowest and most favorable position.

RESULTS OF OPERATION: Statistical figures, according to Ochsner, have shown that subphrenic abscesses treated nonoperatively showed a mortality rate of 91.1 per cent, whereas in the collected cases treated by drainage, the mortality rate was 33.6 per cent. The statistical figures of Ochsner show also an improvement in the results when operating extra-peritoneally. In thirty-one of his cases personally operated upon with the retroperitoneal technic the mortality rate dropped even to 9.7 per cent. The administration of sulfa drugs and penicillin after the operation will lower still more the mortality.

HYDATID DISEASES OF THE LIVER (Fig. 136)

Hydatid cysts of the liver are a matter of urgent surgery (1) in case of suppuration, and (2) in case of rupture.

SUPPURATION. An intrahepatic cyst is in most cases infected from the ducts, but a blood-borne infection must also be assumed in some cases. The most frequent organisms are the *Bacillus coli*, the streptococci and the staphylococci. However, according to Dévé, Goerbet and Delamare, also anaerobic organisms are found as infecting agents.

PATHOLOGY. After the infection, degeneration of the daughter cysts takes place, but sometimes there have been found healthy daughter cysts floating in bile-stained pus. The pus has a fetid odor when infected and is bile-stained; sulphurated hydrogen gas and carbon dioxide are found over the fluid level.

In some cases, despite the large amount of pus collected in these liver cysts, serious symptoms may be absent even when this suppuration has been present for months. In most cases the condition of the patient is a very serious one and there is usually a state of septicemia with jaundice. The symptoms are chills, perspiration, vomiting, intermittent fever, dyspnea and cachexia. Local symptoms depend upon the size of the cyst and its position.

DIAGNOSIS. History and the geographical distribution of a hydatid

disease suggest the likelihood of a right diagnosis; otherwise the case is usually taken for an obstructive jaundice through gallstones followed by septicemia.

When palpating, the circular shape of the dullness in the enlarged liver and also the anaphylactic symptoms such as urticaria, may help

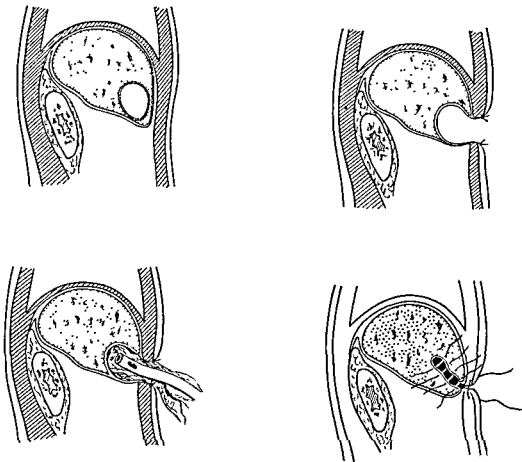


FIG. 136. Surgical management of hydatid cyst of the liver.

some in differentiation. Pylephlebitis with liver abscesses and any other perforation of the intestinal tract may be diagnosed in error. But by means of radiography and immunologic tests the correct diagnosis can be made, according to Devé, in about 90 per cent of the cases.

When not operated upon in due time, cachexia, albuminuria, persistent diarrhea and heart disease may result. In other cases rupture in different places is the outcome.

RUPTURE. A rupture of a hydatid cyst whether it is clean, or infected and suppurating, may occur into all the surrounding organs or into the free peritoneal cavity. Obviously the latter causes the most acute and alarming symptoms. Cases of rupture have been reported into the lungs

and bronchial tree, the alimentary canal, into the stomach, duodenum, ileum, bile ducts with ensuing obstructive jaundice, into the portal vein and into the inferior vena cava.

The history of the patient often gives evidence of trauma as the definite cause of rupture of the cyst. But there are certainly cases without any such accident, especially in the case of suppuration.

INTRAPERITONEAL RUPTURE. Sudden colicky pain followed by peritoneal irritation, meteorism, vomiting, shock, high temperature and the diminishing in size of the formerly enlarged liver are typical signs. Anaphylactic symptoms, especially in the case of the rupture of a non-suppurating cyst, are common, and it has been emphasized that the initial shock may in these cases be an anaphylactic sign rather than due to peritoneal irritation. Eosinophilia up to 50 per cent is very significant. Jaundice through the resorption of bile may follow sometimes very soon. Ascites varies in amount and biliary peritonitis is often diagnosed, especially when tapping the abdominal fluid, which shows an intense bile-stained appearance.

The acute symptoms may disappear and in the chronic or subchronic course a typical clinical aspect may develop which is known under the name of *choleperitoneum hydatideum*. It is characterized by the biliary effusion into the peritoneum with echinococcic cysts which multiply in the course of the disease.

The prognosis is a very bad one, the statistical figures showing a mortality 76 to 90 per cent (Dévé). There is no question that an urgent operation as soon as possible is the best treatment. Small ruptures, i.e., the forming of communications between a hydatid cyst and smaller bile ducts, are very common and therefore a bile-stained fluid is frequent.

But also a rupture into larger bile ducts and the main hepatic duct occasionally takes place. Less frequently ruptures into the gastrointestinal tract occur.

The clinical signs of rupture of a cyst are: severe pain of a colicky nature as in gallstone colic. The patient may be found doubled up, sweating, and vomiting; jaundice is a symptom found in about 80 per cent of the cases.

According to Dupreyen in 23 per cent of the patients a natural cure occurs. In the other cases urgent surgical intervention is necessary.

HOW TO DEAL WITH THE CYST. The following procedures can be applied according to the individual case: (1) Suture of the cyst into the wound followed by drainage (a two-stage operation). (2) Thorough cleaning of the main cyst of all the hydatid elements followed by suture of the cyst. (3) Extirpation of the whole sac, with or without resection of the wedge-shaped piece of the liver.

THE OPEN METHOD. This method is frequently applied as a two-stage operation in cases of nonruptured cysts. The liver surface is sewn into the peritoneum as a first-stage and the cyst opened after ten days with a cautery. In deep-seated cysts this method would, however, be a very tedious procedure, which the poor state of the patient forbids. The thorough cleansing and washing of the cyst especially with formol may be carried out later in a second-stage when the patient has recovered from the first operation.

The postoperative course may be very long especially when the wall of the cyst is already calcified. This calcified, rigid wall may hinder the shrinking and healing of the liver wound, a foul secretion may last very long as well as a bile fistula, which may endanger the life of the patient even long after the operation.

These hepatic bile fistulae are caused either by perforation of the hydatid cyst into larger hepatic bile ducts or by injury to such ducts when removing the hydatids. For this reason one should be careful to avoid any injury to the external capsule formed by the host. The frequency of such long lasting fistulae is given by different authors as occurring between 50 to 75 per cent (Thöle, Partsch, Berger). The mortality depends upon the general state and the amount of the peritoneal infection in case of intraperitoneal rupture.

For the repair of such long lasting bile fistulae many procedures have been described. The best seems to be drainage into the common duct by means of a tube introduced from the opened duct through the liver to the fistula. This rubber drain is removed when the fistula is closed. (See also injuries of bile ducts.)

THE CLOSED METHOD. A primary closure of the cyst during the operation has many advantages and has been carried out by many surgeons (Thornton, König, Bond, Bobrow, Delbet, Posada, etc.). This primary closure is carried out in the following way:

After a thorough removal of the daughter cysts, the flabby cyst wall is folded in to fill the cavity as much as possible. Catgut sutures through the whole cyst and surrounding liver tissue may help the occlusion of the cavity. For obvious reasons only superficial cysts are suitable for this purpose.

No doubt this method may have ideal results, but disadvantages and disturbances in the postoperative course may take place especially when the wall of the cyst is too rigid. The suture is not quite safe in all cases; suppuration and bile leakage may occur.

Several cases are reported in which the suture had to be reopened and secondary drainage performed (five cases reported by Garré; two cases out of fourteen by Goljanitzky; Vegas and Cranwell reported ninety-two cases in which primary closure had been performed, seventy-one of

which healed uneventfully; in sixteen secondary drainage had to be performed and five had fatal outcomes.)

METHOD OF INJECTION OF FORMALIN. Dévé in 1901, advocated this method. He injected 4 per cent formalin into the cyst in order to kill residuant brood capsules and scolices. This method was applied by many surgeons who claimed satisfactory results.

Another method is the filling of the cyst with saline solution. The wall of the cyst is often soft and rather thin. As already mentioned, when a calcareous adventitia exists the opinion of Dévé and Killington that drainage should be avoided when possible should be followed.

The extirpation of the echinococcic cyst with the capsule is certainly the most ideal operation but can be carried out only in a very limited number of cases. These cases are suitable for extirpation when the cyst is connected with the liver only by a peduncle of the liver tissue. Over the cyst the border of the liver lies superficially so that the wedge-shaped resection of the part of the liver containing the cyst can be carried out without great difficulty.

A helpful procedure is the infiltration of the cyst with a $\frac{1}{2}$ per cent saline-adrenalin solution.

In any case the wedge-shaped resection is to be recommended more than the removal of the whole cyst from the liver which is comparable to a classical cholecystectomy. Injury to the liver is greater and serious bleeding from the crushed tissue as well as bile leakage take place.

In the case of multiple echinococcic cysts it is advisable not to be too radical but to perform the radical operation at a second stage.

Electrocoagulation of the inside of the cyst must be considered as a method worth while trying because of its many advantages. A thorough destruction of all hydatids can be achieved deep in all crypts and hidden spaces.

An extirpation of the sac is not necessary. Control of hemorrhage is perfect. We can also safely remove calcareous spots when electrocoagulation is performed. Primary closure with packing the cavity with omentum can be performed in a great many cases to control bleeding.

URGENT OPERATION IN THE CASE OF RUPTURE. The operation should be performed only when the patient has recovered from the first shock, which must be considered not only as a peritoneal shock but also as an anaphylactic one. All means should be applied to combat this shock as quickly as possible. Blood transfusion and the application of calcium have proved helpful as in all similar states.

A rupture into the free abdominal cavity takes place in 14 per cent of all cases according to Dévé's statistical figures. Without operation the prognosis is very bad, the mortality being between 76 and 90 per cent. An early operation is the only hope for lowering the mortality. The

chance for a lasting recovery is nevertheless only 70 per cent according to D  v  .

OPERATION. By a midline or paramedian laparotomy the abdominal cavity is opened. The incision should be large enough to allow a thorough search of the whole abdominal cavity for the cysts. It seems to be wise to deal first with the ruptured cyst before cleansing the abdominal cavity. Therefore, the liver in the region of the ruptured cyst is to be inspected while this field should be walled off from the rest of the peritoneal cavity. A suction apparatus with a wide tube is introduced into the cyst and the contents sucked out. The peritoneal cavity should be guarded by sufficient gauze packs to avoid further contamination with hydatid elements. The use of black packs (D  v  ) has been found advantageous because the white brood capsules or small daughter cysts show up very clearly.

The size of the cyst and the d  bris may cause sometimes a blocking of the tube when it is not large enough. Sometimes the content is so thick that the sucking does not work at all. The cyst is now opened and is dealt with by means of one of the methods already described.

After taking care of the ruptured cyst, the liver has to be thoroughly inspected for other cysts. But if a poor general state exists one should not insist on a radical removal of all of these cysts; it is better to leave these unruptured cysts for a second-stage operation.

All abdominal pads are removed and the cavity thoroughly inspected. All floating cysts are removed and the cavity washed out with warm saline or slightly antiseptic solution.

We should not forget to examine both subdiaphragmatic spaces: over the liver and over the spleen. The gauze padding from the cyst is then removed. When possible the wall of the cyst is folded in and the abdominal wall closed with or without a drain according to the individual case and the conditions.

INTRATHORACIC RUPTURE OF HYDATID CYSTS. The frequency of rupture into the thoracic cavity may find perhaps its explanation by a negative intrathoracic pressure. The diaphragm can be elevated up to the third or second rib, atrophied and thinned by the growing cyst, so that it may penetrate into the intrathoracic cavity. A rupture following such a penetration is very frequent. In most cases suppuration occurs first and precedes rupture.

Different types of rupture have been observed: (1) into the pleural cavity, (2) into the bronchus, and (3) an encysted empyema develops forming an intermediate pouch between the hydatid cyst and the bronchial opening.

COMPLICATIONS. One of the main questions is whether the cyst has been infected or not. The contents of the noninfected cyst may be

aseptic for a long time also in the thoracic cavity, but sooner or later an infection practically always takes place. The daughter cysts burst one after the other, are expectorated and are found in the sputum as fetid bile-strained grapeskin-like masses. Cholepyothorax or cholepneumothorax is common.

CLINICAL ASPECT. The perforation into the intrathoracic cavity is never as dramatic as a rupture into the free abdominal cavity. The operation, therefore, is rarely as urgent as in the case of intraperitoneal rupture.

OPERATIVE PROCEDURE. In some suitable cases a radical operation can be performed for both the intrathoracic and intrahepatic cyst through the transpleural transdiaphragmatic way. After removal of the the intrathoracic or lung cyst the way of communication into the liver is laid bare, the remaining hydatids from the liver are removed and the inside is electrocoagulated. Primary closure should always be attempted.

POSTOPERATIVE COMPLICATIONS. In the case of closure without drainage the patient must be carefully watched and we must be prepared either to aspirate the cyst through a suture line or to reopen the incision partly and to perform a secondary drainage. When drainage has been established, leakage from a persistent sinus may be the source of annoyance and trouble both for the patient and the surgeon. Anaphylactic phenomena in the postoperative course are very frequent; injection of calcium with parathormone is the best measure to prevent and to treat these cases.

GALLSTONE ILEUS

When a large gallstone passes from the gallbladder into the intestinal tract, it may give rise to intestinal obstruction. These patients are usually admitted for operation as cases of emergency under the general diagnosis of "ileus," but the gallstone ileus has not only special pathology but also very characteristic clinical signs and symptoms which enable us to make a correct diagnosis.

This correct diagnosis is useful when we choose our operative procedure. Therefore, it seems justifiable to deal with gallstone ileus separately from other kinds of intestinal obstruction in this consideration of urgent operations on the biliary tract.

Perforation of a large gallstone into the intestines—in most cases into the duodenum—must be considered first. This occurrence may take place only with the clinical signs of severe gallstone colic followed by general relief.

Local peritoneal irritation may be present, but owing to the fact that rupture of the gallbladder into the duodenum is only the last stage of a long pathological process, alarming symptoms of peritonitis may be entirely absent. The perforation takes place as a concealed process, the

gallbladder being adherent to the duodenum with strong fibrous tissue formed over a long period of chronic inflammation.

The formation of such an internal fistula after the perforation of a gallstone from the gallbladder into the intestines is quite frequent. Kehr found them 100 times in 2,000 gallstone operations, and collective statistical figures by Jancke show that in 6,280 operations 103 cases of spontaneous fistula have been found, fifty-nine into the duodenum, thirty-five into the colon and nine into the pyloric part of the stomach. Perforation into the colon rarely leads to intestinal obstruction by the perforating stone. Perforation into the duodenum is the usual case and in rare instances into the pyloric part of the stomach. No evidence is given that a stone causing gallstone ileus has ever passed from the gallbladder into the common duct, through the papilla and into the duodenum.

According to statistical figures gallstone ileus presents about 2 to 6 per cent of intestinal obstruction, but these figures vary considerably in different countries. In the majority of cases the patients are women.

DIAGNOSIS. The history of the disease is most important for the diagnosis. Previous gallstone colic and especially the careful description of the last attack may sometimes put one on the right track. But between the passage of the gallstone through the perforated gallbladder into the duodenum and the appearance of the first signs and symptoms of an ileus a considerable time may elapse, so that a historical reconstruction of the process by questioning the patient may prove difficult at times.

Intestinal hemorrhage and melena, i.e. black tarry stool following gallstone colic is sometimes significant. The patient should be questioned especially as to the appearance of the stool.

SIGNS AND SYMPTOMS. The most characteristic sign of gallstone ileus is the spontaneous improvement with frequent recurrences. In the interval between such attacks of intestinal obstruction, which may last up to twenty-four hours, gas and stool can be passed. The patient feels relieved until another attack sets in. Sometimes these alterations of a total obstruction and the disappearance of all symptoms are not so clearly pronounced. As a rule the symptoms gradually become worse and the periods of relief grow shorter until a complete obstruction has been established.

This form of gallstone ileus may be easily taken for an obstruction caused by a malignant tumor. The obstruction is rarely a mechanical one alone, because it can always be stated when operating that the stone being smaller than the lumen of the loop in which the obstruction took place could easily have passed through the intestines. It is practically always a spasm around the stone which makes the occlusion complete. Based on these facts the clinical symptoms of recurred obstruction and interval of relief find their explanation.

DIFFERENTIAL DIAGNOSIS. The alternation between total obstruction and subsequent relief in a gradually deteriorating course is recognized as a very characteristic clinical sign for gallstone ileus. Nevertheless, the differential diagnosis is sometimes difficult.

The attacks of colicky pain with signs of obstruction and tenderness of the abdomen followed by relief when the patient passes gas may be taken for renal colic. Examination of the urine (red blood cells) in the latter course helps toward a correct diagnosis.

The colicky pains are frequently localized in the right lower abdomen so that the differential diagnosis of acute appendicitis must sometimes be considered. The blood count (leucocytosis in case of appendicitis) may be of diagnostic value.

The diagnosis of gastric or duodenal ulcer must also be taken into consideration at times.

X-RAY EXAMINATION. The x-ray diagnosis of gallstone ileus can be based on the following findings: (1) general diagnosis of an intestinal obstruction, (2) evidence of a gallstone shadow in the intestines, and (3) evidence of an internal biliary fistula.

1. The patient should be examined in an upright or sitting position behind the screen. The presence of multiple horizontal fluid levels in the small intestines with transparent areas of gas over the fluid surface makes the diagnosis of ileus easy and certain at the first sight. When shaking the patient, the movement of the fluid is always very significant. The distended loops hang over the normal ones like wings. The Kerkring plicae are often visible. In some rare cases the ring-shaped shadow of the obstructing stone may be seen proving the presence of a gallstone ileus.

2. The direct evidence of a gallstone is not always present. It is well known that even in the gallbladder gallstones are not always visible, and certainly not a cholesterol stone.

On the other hand, the larger gallstones are frequently pure cholesterol stones or are composed principally of cholesterol. But there are cases reported in which a gallstone lying in the intestines gave a clear shadow, so that the diagnosis of gallstone ileus could be made based on these findings. (Sävenberg.)

3. The evidence of an internal bile fistula as shown by x-ray is very promising because this is a finding independent of the acute stage of obstruction, and when found after an attack of ileus, may be of the greatest help.

An x-ray diagnosis of an internal bile fistula can be made (1) when after a contrast meal the barium is found to ascend into the common duct, producing a picture sometimes even of the intrahepatic duct; (2) through the evidence of air in the gallbladder and bile ducts. Several cases of gallstone ileus have been reported in recent years. (Lönnerblad.)

Petrén reports nine cases of gallstone ileus all of which had been x-rayed. In none of them could a gallstone be found in the x-ray picture, but in five of these cases a strip-shaped collection of gas was seen in the common duct or gallbladder indicating the internal bile fistula.

The question must be asked: Is the collection of gas or of barium meal in the bile ducts certain evidence of a spontaneous internal bile fistula? It is certain that the duodenal content may also slip through an open papilla into the common duct, especially when a large stone previously has passed through. Another possibility is that in the case of cholangitis the gas found in the common duct may be produced by bacteria.

However, it can be said that these are rather exceptions and that in the great majority the collection of gas or of barium meal in the bile ducts can be considered as evidence of an internal bile fistula.

I quite agree with Borman and Rigler when saying: "Roentgen examination of the gallbladder region as well as of the whole abdomen in case of suspected intestinal obstruction may reveal evidence of a biliary fistula and thus make the origin of the obstruction clear. This type of examination should be made in all cases having symptoms of obstruction of obscure origin."

TECHNIC. The patient is x-rayed over the Potter-Bucky frame. The collection of air can be enlarged when the patient is asked to swallow air several times and this air is distributed by massage over the stomach into the duodenum. In order to fill the bile ducts with barium meal the patient lies down on his right side before being x-rayed with pelvis lifted.

TREATMENT. There can be no doubt that sometimes, when applying conservative treatment with antispasmodics, like belladonna, papaverin, etc., the spasm around the stone may give way so that the acute obstruction may subside. A stone then may even be passed with the stools. But when trying such conservative treatment and waiting for a fortunate outcome we must realize that this waiting includes a considerable amount of danger. Antispasmodic medication may relieve the alarming symptoms for a certain time but the intestinal wall may already be damaged and even necrotic, thus involving the danger of rupture. Later we would have to operate under unfavorable conditions. I think when the diagnosis of an intermittent intestinal obstruction caused by gallstone has been made, we should not postpone the operative procedure, which is quite favorable when performed under good general conditions.

The operative mortality has formerly been very high, between 33 and 92 per cent (Wagner). Failure of making a correct diagnosis and a too lengthy postponement of the operation are the main reasons for the high mortality. Early diagnosis and early operation lead to better results. In thirteen operations Petrén had five patients cured and eight with fatal results. All seven patients operated upon between 1910 and 1924 died,

while five of six patients survived who were operated upon between 1928 and 1938. Petrén emphasizes that the improvement in results is certainly due to early diagnosis and early operation. Wakeley and Willway operated successfully upon eight out of eleven patients.

X-ray diagnosis must be considered a great help for the diagnosis of gallstone ileus. Evidence of an internal bile fistula, i.e., communication between gallbladder and duodenum, can be given by x-ray examination in an interval between two attacks.

TECHNIC: INCISIONS. A right, lateral muscle-splitting incision at McBurney's point is advantageous. This incision should not be too small. The chance of opening the peritoneal cavity right over the critical spot is very good, and the advantage of limiting the extent of the operation in the abdominal cavity should certainly be attempted in any way possible. When we are forced to search the whole intestine, we should do it in a systematic way upward from the collapsed loops or, as a rule, take the ileocecum as a landmark for a search in both directions.

I am in favor of putting back immediately any loop which has been investigated. The immediate eventration undoubtedly facilitates the operation for the surgeon but it increases the operative risk (shock, spreading of infection). While eventration in another kind of ileus can hardly be avoided, it can be avoided in most cases of gallstone ileus. This lateral incision over the ileocecal region also has the advantage of being extended for an exploration of the biliary system and the gallbladder, if necessary; and in case we should be forced to perform a cecostomy as an emergency, this incision has its advantages. From the technical point of view it is advisable before making an incision through the intestinal wall over the stone to move the stone from the spot of obstruction to another part, no matter in what direction, so that we can incise and suture at a spot where the blood supply of the wall is not damaged.

Before opening the intestinal loop we should always wall it off carefully from the abdominal cavity and the abdominal wall should be carefully protected, the content of the obstructing loop being very infective. Any leakage and contamination should, therefore, be avoided.

The loop to be opened should be clamped and the content between the two clamps around the stone carefully aspirated by means of a syringe. A cleansing also by means of a double way syringe must be considered as useful to avoid infection. Contamination of the abdominal wall may be followed by a long lasting necrotizing cellulitis. When there is any doubt about the blood supply or if there is suspicion of necrosis of the wall, it is much better to perform a resection of the bowel. Enterotomy should be performed only in advanced cases when the condition of the patient is very poor. Postoperative administration of penicillin and sulfa drugs should also be resorted to, if there is a rise of temperature.

ACUTE CONDITION OF THE GALLBLADDER

Under this term is understood the acute obstruction of the gallbladder with all its complications. It includes also acute cholecystitis. Recent advances in pathology and surgery have proved to us that the usual form of classification of acute cholecystitis is not quite satisfactory from the practical point of view.

What we usually call with the popular name, "acute cholecystitis," is in fact an acute flaring up of latent or chronic infection of the gallbladder, which follows the sudden check of bile outflow caused by an acute obstruction in most cases by a stone blocking the cystic duct.

In addition to this, the impacted stone may cause an interference with the blood supply, resulting in a check of the venous reflux and hemorrhagic infarction. This causes serious damage to the wall of the bladder, producing wide partial necrosis and leading to rupture or at least to the formation of deep ulcers in the case of purulent infection.

There have been some cases reported in which this hemorrhagic infarction and partial or total necrosis of the wall was the real and only pathological change in the gallbladder, while one could hardly speak of an acute cholecystitis in the bacteriological sense. Not only the bile but also the gallbladder has been found sterile and the pathological changes were only those of a hemorrhagic infarction and not inflammatory changes.

In rare cases the obstruction was found to be caused by an axis torsion of the abnormally movable bladder, pedunculated only on the duct and the cystic artery with a small fibrous band connecting the bladder with the liver bed.

But in all these cases the characteristic abdominal accident consists in acute obstruction, gallbladder ileus, as we may call it. When seeing the patient first we cannot and should not make any other diagnosis than that of acute obstruction of the cystic duct. The differential diagnosis of an acute cholecystitis or an acute hemorrhagic infarction following the obstruction can be made only when operating. It is worth while mentioning also that even by x-ray we cannot make any other diagnosis than that of obstruction of the cystic duct.

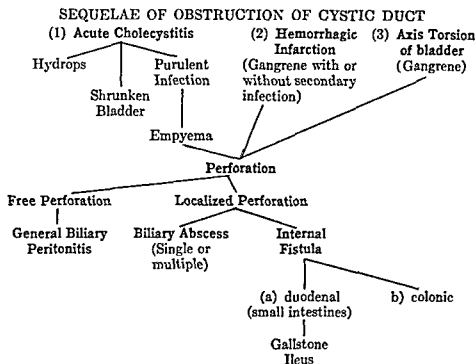
We should bear in mind that in practically all cases of so-called acute cholecystitis the acute obstruction of the cystic duct or of the neck of the bladder with its sequelae, namely, the check of the outflow and drainage of bile and the damage to the gallbladder wall, are the main pathological points; all other signs and symptoms, including possible complications, can be easily explained and understood.

For obvious reasons the pathologist is rarely in a position to acknowledge the importance of the **acute obstruction** in all cases of acute cholecystitis. He examines only the excised bladder; no spasm can be found

any more, the stones may lie loose in the bladder and the vital importance of the obstruction may not be as evident in the specimen as it is when operating *in vivo*. But I can scarcely remember one single case of acute cholecystitis in which this factor of obstruction whether by stone, mucus or fibrin has not been evident.

What I would like to emphasize here is that considering all the possible consequences, the acute condition of the gallbladder may become at any time a matter of urgent surgery and must be discussed fully from clinical and pathological points of view.

The following table gives a classification of the aspect of the acute condition of the gallbladder and its possible complications:



The pathological changes in the bile and the mucous membrane of the wall after such an acute obstruction of the cystic duct depend on various factors: the kind of virulence of the different bacteria, the reaction of the tissue and the general resistance.

PATHOLOGY. The pathological changes in the gallbladder after an acute obstruction may be described as follows:

The golden-brown color of the bile (bilirubin) changes through stasis into green by an oxydation process. The germs multiply and the cells of the mucous membrane react in producing mucus and fibrin. Leucocytes immigrate into the bile, which becomes turbid, and the amount of fluid increases considerably in a short time. The outflow being checked, the gallbladder distends and the initial acute colicky pain, indicating the

obstruction by a stone, is followed by a rather permanent feeling of pressure due to the distention of the bladder.

Meanwhile not only an abundant secretion takes place from the mucous membrane into the gallbladder, but also a resorption, causing symptoms of general illness according to the virulence of infection: increase of leucocytes in the blood, fever and sometimes chills are observed.

The obstruction may cease, especially when a spasm relaxes which has held the stone in its position in the neck of the bladder, and the acute symptoms may disappear. The stone may fall back into the fundus and the infectious content of the bladder may drain again through the cystic and common ducts into the duodenum.

A certain amount of healing of the inflamed gallbladder wall takes place (formation of scars in the necrotic parts, adhesions with the surrounding organs, pericholecystitis) until another obstruction causes a new attack with recurring inflammation. But even when the obstruction persists, the inflammatory process may calm down. The virulence of the bacteria decreases; the bile may even become sterile, the secretion stops, the tension in the bladder relaxes through a certain amount of resorption and the general symptoms of illness disappear.

When the obstruction persists for a longer time, some definite characteristic changes occur in the bladder.

HYDROPS. Through the ferments of the leucocytes, the bile dyes may be discolored, the content of the bladder become clear and a so-called hydrops of the bladder may result.

In the wall of the bladder some changes are observed: a certain amount of atrophy in the mucous membrane, atrophy of the muscular element, increase of fibrous tissue, formation of scars and thickening of serous layers.

Clinically the patient feels a permanent pressure in the epigastrium and the right hypochondrium; the gallbladder can be palpated due to its enlarged size; in some patients definite signs of the lack of gallbladder function appear, namely, decreased tolerance to some food, especially fatty food, constipation, flatulence and belching.

A hydrops of the bladder with its clear, mucous content and thickened wall may be considered as a chronic and rather benign final state of gallbladder ileus which rarely requires urgent surgery. Clinical signs may be absent or limited to a slight chronic pressure or symptoms of the loss of function.

SHRUNKEN BLADDER. Another final state of the permanent obstruction of the gallbladder is the so-called shrunken bladder. The liquid content may become nonvirulent and even sterile and reabsorbed. Atrophy of the mucous membrane takes place; the submucous fibrous tissue shrinks to the size of a walnut and even a hazelnut, so that a stone

fills the whole lumen of the bladder. A few drops of turbid mucus may remain. This shrunken bladder may be considered to a certain degree a process of spontaneous healing.

Things are different when the infection in the obstructed bladder remains undiminished and the content becomes more and more purulent. An empyema develops.

In these cases with purulent content the formation of ulcers and abscesses in the bladder is found very common; they grow in all parts of the wall, the liver bed especially is a favored spot for abscesses reaching deep into the liver tissue. The whole wall of the bladder may become affected by a phlegmonous inflammation with multiple abscesses which may spread into the different surrounding tissue and organs, even leading to general septicemia.

In most cases of this acute cholecystitis the obstruction has been caused by a stone impacted in the neck of the bladder or in the cystic duct. But there are other cases of virulent, acute inflammation in which the normal drainage of the bladder is apparently checked by the swelling of the mucous membrane. This check of outflow may be increased by a kink in the cystic duct following the inflammatory distention of the bladder.

Acute infection of the bladder is observed in the course of different infectious diseases such as scarlet fever, erysipelas, puerperal septicemia, pneumonia and especially in infections with bacilli of typhoid and paratyphoid fever.

In an epidemic during the first World War I have observed eighty-nine cases of such acute cholecystitis in a rather short time. I had to operate upon most of these patients as emergencies, some with a ruptured gall-bladder with biliary peritonitis, others shortly before perforation. From the bile it was possible to obtain a pure culture of typhoid or paratyphoid bacilli.

HEMORRHAGIC INFARCTION. I would like to emphasize that a hemorrhagic infarction as the basis for damage of the wall and cause for rupture is much more frequently found than would appear from the special reports in the literature. There are cases reported in which not only the hemorrhagic infarction of the wall was the outstanding pathological sign but also the more or less serious bleeding into the abdominal cavity after rupture.

The following is a case recently reported by Mailer:

The patient, a sixty-five year old man, had been in excellent health up to a week before admission. He then began to suffer from vague abdominal pain without any accurate localization. On the day previous to admission the pain in the lower portion of the abdomen became more severe; he vomited a quantity of brownish material. On examination the abdomen was found slightly distended and tender in the lower half. There was no response to enemas. Laparotomy was decided upon.

On opening the peritoneal cavity blood welled out and on further exploration it was found that the abdominal cavity contained between three and four pints of partly fluid and partly clotted blood. The source of bleeding was found to be the gallbladder which had a rent about one inch long in the fundus near the point of reflection of the peritoneum from the liver on to its upper surface. Oozing of the blood was still taking place from the ruptured margin of the rent adjoining the liver. There was a solitary large stone in the gallbladder, which was removed. A tube was pushed through the rent and the gallbladder packed with gauze. The patient died three hours later.

The postmortem examination showed that the entire wall of the gallbladder was thickened and hemorrhagic. Toward the neck of the gallbladder the mucosa presented a shaggy appearance. A linear perforation one inch long was present in the fundus close to the hepatic surface.

Cultures taken under aseptic conditions from the content of the gallbladder at the commencement of the examination were found to be sterile.

Microscopical examination revealed that in sections approaching the fundus, capillary and venous congestion became more intense until finally toward the region of the tear the subserous and fibrous muscular coats were massively infiltrated with blood no longer contained within the vessels but permeating the entire wall of the gallbladder like a hemorrhagic infarct. The striking feature was that the degree of inflammatory reaction present in the section was very slight and the appearances were much more those of a circulatory disturbance.

Similar cases are reported by Leared, Schnyder, Gyellerup, Waters and Bartlett.

These cases of extensive hemorrhagic infarction with rupture and bleeding into the peritoneal cavity are rather rare, but microscopical examination of the excised gallbladders have convinced me for several years that vascular disturbance and interference with the blood supply in the case of stone obstruction in the fundus of the gallbladder are much more frequent than is generally believed. This circulatory disturbance has its maximum effect upon the capillaries of the fundus. A rising tension in the gallbladder following the obstruction interferes also with the venous return from the gallbladder to the liver, thus increasing the circular disturbance. This primary damage to the wall through circular disturbance must be considered in many cases as the primary lesion, while infection and local inflammation with formation of ulcers in the damaged wall must be considered as a secondary lesion.

This conception satisfactorily explains the striking feature that in spite of serious necrotizing inflammation the wall of the damaged bladder has been found so frequently sterile.

Here we cannot obviously speak of an acute cholecystitis in spite of the fact that all clinical and pathological signs appear to be identical.

The process is comparable rather to the hemorrhagic infarction in an intestinal loop following a thrombosis of a mesenteric vein.

Denton and Feinblatt have found intramural edema in venous congestion in milder cases and hemorrhagic infarction and gangrene as features of the severe types. Aschoff also mentions hemorrhages in his monograph but is rather inclined to attribute them secondarily to the infection.

All these pathological facts support the opinion mentioned above that the obstruction of the cystic duct is the primary and most important one. This acute obstruction is the real meaning of the so-called acute condition of the gallbladder as a matter for urgent surgery.

AXIS TORSION OF THE GALLBLADDER. This form of an acute obstruction is somewhat rare and is due to an anatomical abnormality. It can occur only when the gallbladder, entirely covered by serosa, is freely movable and only pedunculated on the cystic duct or connected with the liver by a band near the neck of the bladder. For obvious reasons, in such a pedunculated bladder an axis torsion may easily occur, followed by acute obstruction of the outflow of bile and a checking of the blood supply. Gangrene and rupture may quickly ensue. Several cases are reported in the literature.

Trauma and suddenly increased pressure on the abdominal wall are sometimes given as causes but in other cases no accidental reason can be given at all; the patient has an acute abdominal attack, which is usually taken as gallstone colic or acute appendicitis.

A clinical diagnosis has, therefore, never been made in any of the few cases reported in the literature, but the indication for an urgent operation is always given. Huddy has reported the case of a woman of seventy-one whom he operated upon for a lump which he believed to be an appendicular abscess. The lump proved to be the gallbladder which had made one turn on its neck in an anticlockwise direction. It was hanging from the liver edge and only a narrow mesentery was found in the region between the liver and the neck of the bladder. After removal of the twisted gallbladder, which could be performed very easily, the patient recovered. In another case, described by Irving, the gallbladder had undergone a complete rotation, the twist being not in the cystic duct but in the gallbladder itself.

When operating at an early stage, before gangrene and peritonitis have set in, the prognosis is favorable. The operation consists in removal of the bladder. From the technical point of view the operation is easy and can be performed very quickly owing to the fact that the organ is freely movable, pedunculated only on the cystic artery and the cystic duct and not fixed to the liver bed. After simple ligation of the pedicle, the organ can be removed. No drainage is necessary, if perforation and peritonitis have not taken place, but a covering of the stump with

peritoneal tissue from the hepatoduodenal ligament is always advisable.

CLINICAL SIGNS OF GALLBLADDER OBSTRUCTION. The patient is taken ill with a severe colicky, doubling-up pain in the right upper portion of the abdomen. Occasionally, cold drinks or a fatty meal are mentioned as the cause. At other times the attack takes place in the middle of the night, when no cause for the sudden obstruction can be found. Chill is observed but not in all cases. The region of the bladder is very tender, muscular rigidity is pronounced, no jaundice follows the attack and fever is frequent. The increase of leucocytes depends on the degree and kind of the infection. The peritoneal irritation may remain as it is after the first attack, or may spread downward, a valuable sign that the case is urgent.

In some instances acute obstruction takes place with less dramatic symptoms. The patient complains only of the very characteristic pressure on the epigastrium, "cystic duct symptom," sometimes piercing through to the back. Pains in the shoulder are observed in a good number of cases. The right phrenic nerve is generally more tender than the left one when pressed on the outer border of the sternocleidomastoid muscle. All other clinical signs depend on further developments: the spreading of infection with formation of abscesses, or rupture with general or localized peritonitis.

The most dangerous accident following the obstruction of the gallbladder is necrosis leading to rupture. This may follow the acute obstruction in a very short time, between twelve to twenty-four hours. This happens in the majority of cases when the acute infection has been of recent date and the wall of the bladder has not been thickened after previous attacks. In my own cases of such bladder perforation into the free abdomen the wall was rather thin without any adhesions.

Cholecystography has proved of outstanding value for all gallbladder diseases, especially for the obstruction of the cystic duct. The dye (tetraiodophenolphthalein) given orally or intravenously is secreted by the liver cells with the bile and can reach the gallbladder only through the cystic duct. The appearance of a gallbladder shadow between fourteen and eighteen hours after application of the dye is, provided a correct technic has been employed, so reliable that we may conclude from the absence of a gallbladder shadow in a series of x-ray pictures that the cystic duct must be obstructed in some way.

It can be said that nowadays the diagnosis of obstruction of the cystic duct is based chiefly on the x-ray examination.

In the very acute stage of an obstruction we certainly do not perform a cholecystography. But when the acute symptoms disappear quickly so that we can postpone the operation, it is always wise to confirm the clinical diagnosis by cholecystography.

CLINICAL SIGNS OF RUPTURE OF THE GALLBLADDER. It is sometimes

very characteristic that in case of rupture of the bladder the patient feels relief, as is usually the case when an organ or an abscess under heavy tension bursts. Therefore, the typical painless interval between rupture and appearance of the sign of general peritonitis sometimes arouses dangerous optimism in the observer.

The signs of general peritoneal irritation start in the right side of the abdomen. The tenderness and rigidity of muscles can be more pronounced occasionally in the lower portion of the abdomen causing great difficulty in distinguishing this condition from a perforated appendix. Tenderness in the intercostal space downward from the eighth rib, in the side and behind is sometimes significant. Dullness follows in the whole right side of the abdomen due to the liquid content. The symptoms of general peritonitis, however, sometime develop very quickly. The bowels become distended, no flatus passes and the rate of respiration increases; but characteristically the pulse rate may be low in contrast to the serious general aspect (result of reabsorption of bile salts; see injuries).

Fluid in the peritoneal cavity can be diagnosed in the very first hours, much earlier than would be the case if this fluid were caused by inflammation. Skin and conjunctivae become stained slightly yellow. In the urine bile dyes are found after a few hours.

The differential diagnosis from that of perforation of a gastroduodenal ulcer or an acute appendicitis may sometimes be very difficult. In these cases of gallbladder perforation, as before mentioned, the acute pain relaxes practically immediately after the perforation, while in the case of gastric perforation relief after the rupture is rare and the extreme rigidity of the abdominal muscles in the epigastrium is very significant. The disappearance of the dullness over the liver, caused by the presence of air in the abdominal cavity between the diaphragm and convexity of the liver, can easily be seen on the x-ray screen with the patient in an erect position.

The differential diagnosis between gallbladder perforation and the perforation of a high appendix may in earlier stages prove difficult and even impossible. One must be satisfied with the general diagnosis-perforation—and operate only with that point of view in mind.

LOCALIZED PERFORATION. When because of former attacks of inflammation extensive adhesions (pericholecystitis) have been formed between the gallbladder and surrounding organs, such as the duodenum, colon, omentum or anterior abdominal wall, the content of a ruptured bladder will empty not into the free abdominal cavity but into the spaces between the adherent organs, thus forming a localized biliary abscess.

Stones may also empty into this abscess which may spread down-

ward along the ascending colon, causing more or less widespread infection in the abdominal cavity. In other cases the abscess may perforate into the subphrenic spaces, causing subphrenic abscesses; or it may even perforate into the abdominal wall and cause a cellulitis there.

The clinical signs are less stormy than in the case of free perforation. Usually a severe colic precedes perforation. But muscle rigidity is very pronounced and much more extensive than in ordinary colic. It sometimes embraces the whole right portion of the abdomen. Temperature is high; leucocytes are found up to as high as 20,000, and bile dyes are found in the urine. The persistent and spreading rigidity of the abdominal muscles and the undiminished or even increasing tenderness lead to the right diagnosis.

A purulent gallbladder in a state of acute obstruction may perforate into the adherent duodenum or colon, causing a spontaneous fistula. When perforating into the colon, a serious septic state may result because fecal masses from the colon pass through the fistula into the gallbladder, there causing a fecal cellulitis with wide necrosis and abscesses in the liver.

These patients develop a serious condition of septicemia with alternating temperature, chills and even jaundice. Such a case will be reported later when describing the electrosurgical operation, which gives the most satisfactory results in these instances.

Stones perforating through such a fistula into the duodenum may be very large at times and give rise to gallstone ileus.

SURGICAL INDICATIONS. I have discussed these sequelae to an acute obstruction of the bladder fully because they are of outstanding importance for surgical indications, i.e., to what degree this condition must be considered a matter of urgent surgery.

Now the following questions must be answered: What is the frequency of perforation? How frequently does general peritonitis follow gallbladder perforation? How is the mortality rate affected under conservative treatment and under delayed operation?

Recent statistical figures on a larger scale present quite a different picture to what has been commonly believed.

First, it was said that the danger of perforation in acute cholecystitis is very small and can by no means be compared with the danger of perforation in acute appendicitis. On the other hand, when perforation takes place the danger of general peritonitis is much less than in acute appendicitis. Therefore, conservative treatment is quite justified and operation can be considered in practically all cases as in interval operation.

I mention a few statistical figures of recent times: In a series of 474 cases, collected by R. Franklin Carter, Carl H. Green and John Russel

Twiss, there were forty-six perforations, i.e., 9.7 per cent took place under conservative treatment. The mortality rate was 19.5 per cent; at subsequent operations the morbidity was very high. Seventy-seven per cent of all cases of perforation had peritonitis at the time of operation, 60 per cent of whom had a diffuse or generalized peritonitis, while 40 per cent were localized by the omentum and colon.

The high incidence of peritonitis in an acute perforation has been emphasized and substantiated by the findings of Heuer, Boyce, Veal and MacFetridge. They emphasize the double menace of diffuse peritonitis in acute cholecystitis.

I cannot go into details here on the valuable and most interesting evidence given in statistical figures by these authors. But, as they say, it is reliable evidence against the teaching of "watchful waiting."

Their conclusions are:

(1) Perforation of the bladder during an acute attack is as frequent and as fatal, judging from statistical reviews, as they found it in acute appendicitis.

(2) Perforated cholecystitis in our cases did not localize as frequently as has been reported in other series.

(3) Watchful waiting is not a safe method.

There can be no doubt that in recent times surgeons with special experience in gallbladder surgery have come to accept more and more the opinion that there is danger in any delay. This is not surprising for any one who has tried to follow carefully the pathological changes resulting from an acute obstruction. It is rather more surprising that it took such a long time for the danger of undue delay to be realized. One reason is that clinical symptoms give no clear evidence as to what actually happens in the abdomen. Even in the case of gangrene following the obstruction symptoms may entirely disappear after the attack. Sometimes the attack may itself be a very slight one.

Cholecystography giving evidence of obstruction of the cystic duct ushering in serious damage to the wall has proved of special value in these cases. I quote briefly the following case, from the year 1924, the first year of cholecystography:

A patient complained of sensation of pressure in the epigastrium and undefined signs of stomach-gallbladder trouble. Cholecystography gave evidence of an occlusion of the cystic duct. The clinical symptoms being very slight, I could not make up my mind to perform the operation and decided to make another cholecystographic study a week later. Again total obstruction of the cystic duct was found and I decided to operate upon the patient in spite of his unalarming symptoms. A partial necrotic gallbladder filled with stones was found in *statu perforationis*.

The clinical signs of peritoneal irritation would certainly have ap-

peared on the next or following days, but the most favorable time for the operation would then have been missed.

Heuer has studied 1,565 cases of his own, and in addition 35,000 cases of gallbladder disease gathered from recent literature. He states that gangrene and perforation take place in 20 per cent of all cases of so-called acute cholecystitis when *not* operated upon. Similar figures are given by Kumath: Among 103 cases 23 per cent were perforated. Furthermore, complications arising from an unoperated acute cholecystitis are responsible for a mortality of 20 to 40 per cent under conservative treatment. These figures, based on a thorough study of numerous statistics, give a different picture from what was usually believed, namely, that there is a definite tendency toward healing in acute cholecystitis or at least toward reaching a rather harmless state in which operation may or may not be considered. Valuable statistical figures from different points of view, and answering many of the most burning questions, have been published by Heyd and Hotz. We must state that the majority of surgeons, especially in America, have become more radical and now advocate early operation as a routine. I quote H. Graham: "The time has come when one must justify any delay in removing the acutely inflamed gallbladder." And I quote another sentence from C. G. Heyd: "Teachers of surgery who lend their prestige and give support to a policy of waiting, provide authority for timid surgeons, inexperienced operators and procrastinating practitioners."

My own point of view I may state as follows: The pathological changes discussed evidently show that what we have previously called an interval is sometimes not a real interval. Clinical signs may disappear, but the process advances in the majority of cases, giving rise to a considerable percentage of serious complications that endanger the life of the patient and influence the result of the operation, which we are finally compelled to perform under less favorable conditions. To wait for the chance of a so-called real interval, which may or may not come, is a gamble for which we cannot undertake the responsibility, when we realize the fact that the statistical figures prove that the wall of the bladder is in 25 per cent of these cases damaged to such a degree that necrosis, free or localized perforation, or spreading infection takes place. Therefore, I believe we should consider the **acute obstruction of the gallbladder**, **gallbladder-ileus**, **acute cholecystitis**, or whatever we may call it, a matter of **urgent surgery** in all cases.

However, I am not in favor of **immediate** operation after the attack. The reason is the same as that discussed under cases of abdominal injuries, namely, that we should avoid operating on a patient while in a state of shock, if possible. All intra-abdominal accidents—and as such we must also consider acute obstruction of the cystic duct—imply local

shock as well as general shock to the tissue, thus damaging the biological defense. The signs of such shock, however, subside in twenty-four to thirty-six hours. We can wait this length of time, carefully watching the patient to see whether the abdominal symptoms tend to diminish or become more aggravated. An icebag is put over the bladder and general preoperative treatment should start according to the special case: intravenous injection of hypertonic 50 per cent glucose for the acute edema of the liver which is practically always present in these cases; injection of calcium and parathormone because of the increased tendency to bleed during the acute attack. Meanwhile the bile pigments in the blood should be estimated. After thirty-six to forty-eight hours the patient should be operated upon provided no free perforation calls for immediate intervention.

Technic of Operative Procedures

The aim of the operation for acute obstruction of the gallbladder is (1) to empty and drain the obstructed bladder and thus prevent necrosis, rupture and all the afore-mentioned consequences; (2) to cure the disease radically. Obviously, when operating upon the patient we should do our best to achieve the aim of point two, i.e., to cure the patient by means of the operation, and not only to obviate the immediate danger to life. The aim mentioned in point one can be achieved by cholecystostomy, which is here described.

CHOLECYSTOSTOMY. Cholecystostomy is the oldest operation in gallstone surgery (see comments) and was already mentioned in the Middle Ages when a surgeon opened a swelling of the right upper abdominal wall on the supposition that it was an abscess. Instead of the expected simple pus he found a thick, bile-stained purulent liquid and gallstones. J. L. Petit was the first deliberately to perform a cholecystostomy in 1743 for the removal of stones. Thudichum has described a two-stage cholecystostomy, the first stage consisting in suturing the gallbladder to the abdominal wall in order to open it more safely in the second stage. Kocher was the first to perform a planned cholecystostomy in 1878.

Technic. The operation can be performed easily under local anesthesia with the addition of gas-oxygen. On the operating table the patient is placed in the position of artificial lordosis so as to bring the liver to the surface and thus facilitate exposure.

Incision. The incision should be small. In case of a pure cholecystostomy is performed, the choice of the incision is not difficult because in most cases the gallbladder is easily felt as a lump through the abdominal wall, and a transverse or midrectus incision should be performed right over the fundus of the gallbladder.

After having opened the abdominal wall, the gallbladder appears either freely movable or adherent to the surroundings, and sometimes

even entirely covered with omentum. In case the gallbladder is free, it should be "walled off" from the abdominal cavity with moistened laparotomy pads. If loops of the small intestines appear in the operative

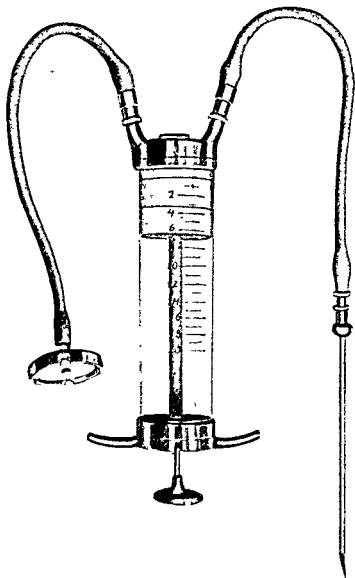


FIG. 137. Two-way syringe.

field, Spivack suggests first spreading the omentum carefully over the small bowels and then inserting the laparotomy pads. This very useful procedure eliminates the troublesome feature of the small bowels appearing in the operative field.

The gallbladder being under tension, it is wise in most cases first to aspirate the liquid content by means of a two-way syringe (Fig. 137). This can be done safely without a drop pouring out of the puncture. The

gallbladder can be washed out with a slightly antiseptic solution by means of the same syringe (Fig. 138). Before removing the needle, the wall around the opening is grasped by a forceps and pulled through a small hole of a moistened laparotomy sponge. The assistant grasps the gallbladder between two forceps and the wall is snipped with scissors

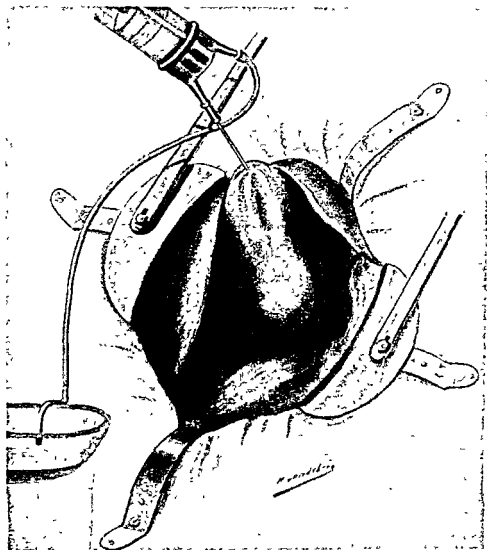


FIG. 138. Aspiration of the gall bladder

wide enough to introduce the forefinger. With this finger the inner coat of the gallbladder is thoroughly searched. This procedure is essential and I do not think it is advisable to introduce a forceps only for removal of the stones. With the finger we have to satisfy ourselves that all stones are removed from the bladder; the liver bed should also be carefully

searched for any abscesses in which stones may be easily hidden (Fig. 139).

It is also advisable to make sure that fresh, brown bile empties through the cystic duct into the bladder as a sign that the cystic duct is free.



FIG. 139. Stone perforating into the liver bed.

When the cystic duct is still obstructed, we should slip two fingers along the outside wall of the bladder, try to draw the stones from the cystic duct back into the bladder and then remove them. When stones are overlooked in the cystic duct, they may either slip into the common duct, causing manifold and even fatal complications in the later course, or when remaining impacted may give rise to a fistula.

After having emptied the gallbladder of all stones, a rubber tube of about one-half inch diameter is introduced into the gallbladder and fixed there by a purse-string suture. The end of this long, catgut thread is knotted around the rubber tube in order to keep it in position. The mucous membrane should be entirely inverted. This procedure prevents to a certain degree postoperative fistulae (Fig. 140).

The further procedure depends on the individual case. We may introduce a simple rubber drain along and outside the gallbladder or, where

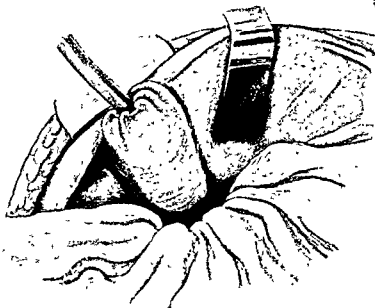


FIG. 140. Cholecystostomy. Insertion of a rubber tube.

there is a badly inflamed wall, it may even be advisable to place a small pad instead of a rubber tube. A suture of the gallbladder to the peritoneum is seldom advisable. When it is performed, care should be taken to avoid tension. When a pad has been inserted, it should not be loosened or removed before the eighth or tenth day.

Comments on the Operation. Cholecystostomy was the routine operation before cholecystectomy was introduced in 1882. It has been gradually abandoned by surgeons except in cases of emergency. It has been revived again especially by Rovsing not only for emergency operations but also in order to preserve the functioning organ. It must be stated quite definitely that these attempts have proved failures. Cholecystostomy cannot restore the function in an inflamed gallbladder as x-ray examinations have shown. It is no use performing a cholecystostomy with this in view.

It must be admitted that in some cases, especially in acute empyema with only one obstructing stone, cholecystostomy has sometimes good and lasting results; but as a whole we must say that the idea that cholecystostomy is a harmless operation in serious cases and has a low mortality rate, is obviously not quite correct. DeCourey found a mortality of 28.5 per cent of the cases and Heyd and Hotz, analyzing 3,080 operations found a mortality rate of 28 per cent in acute cholecystitis. The cause of death is hardly ever the operation itself but rather the uncontrolled and unremoved pathological changes such as abscesses in the liver bed, a phlegmonous inflammation of the wall which spreads in spite of the operation, stones left in the common duct, consequent cholangitis, and others.

I have personally abandoned cholecystostomy and have not performed it since 1926. My main objections to this operation are the following:

1. When opening and draining the gallbladder only, and even when removing any stones through the cholecystostomy opening, the phlegmonous inflammatory process of the gallbladder is by no means controlled in all cases. We frequently find abscesses penetrating not only deep into the liver bed, but also into the surrounding of the neck of the bladder, particularly when stones are lodged there. Such abscesses may easily be overlooked when performing a simple cholecystostomy. I was frequently able to convince myself of such findings and came to the conclusion that a cholecystostomy would have been utterly insufficient in such cases. This fact alone is sufficient explanation for the high mortality.

2. Another serious objection is the fact that we never know for certain whether there are not at the same time concretions in the common duct. A purulent cholangitis may be concealed under the dramatic symptoms of acute gallbladder disease, and cholecystostomy may in this case relieve only one part of the disease, and in some cases not even the most urgent one.

I think it will be in order to mention a case which illustrates this fact and which has been the turning point for me in rejecting cholecystostomy:

A man, fifty-four years of age, in a good general state, had a serious gall-stone attack, lasting a few hours. The gallbladder was palpable and conservative treatment was carried out for a week. Temperature remained subfebrile between 99 and 100° F. Another attack with aggravating symptoms forced the surgeon to perform an urgent operation. Cholecystostomy was done and the mucous membrane of the gallbladder was found partly necrotic and loose in a turbid but not purulent bile-stained fluid. A stone was removed, which was thought to be a solitary one.

The recovery of the patient was very satisfactory in the first week, but soon chills and jaundice set in. A septic cholangitis developed. A

second operation had to be performed, but the patient died as a sequel to septicemia following the septic cholangitis.

No postmortem examination was performed but the clinical picture gave quite a clear idea as to the pathological event.

This operation was performed by a very experienced surgeon who was strongly in favor of cholecystostomy.

The fact that when performing cholecystostomy we cannot satisfy ourselves about the whole underlying pathological process, seems to be one of the greatest disadvantages of the operation. On the other hand, even thinking of cholecystostomy as a primary operation only and having in mind the performance of a radical operation at a second stage, we must say that we have no guarantee, as shown in the above mentioned case, that the second operation will be performed under more favorable conditions.

McKenty, referring to a case in which he had performed cholecystostomy in the first stage and a radical operation at a second, says: "My refusal to do a cholecystectomy during an acute attack on her first admission was a serious blunder, involving dangerous morbidity."

Summarizing I should like to express my personal opinion, that owing to the progress of the technic of a radical operation, especially the electrosurgical operation, cholecystostomy is justified only in very exceptional cases. I mean cases which are determined more by general conditions such as extreme old age of the patient and complications arising from other diseases (heart failure, serious bronchitis, pneumonia, etc.) or surrounding conditions such as the absence of an experienced surgeon or the necessary surgical equipment for a radical operation.

Furthermore, cholecystostomy may be justified when the acute obstruction of the gallbladder is combined with acute pancreatitis. An extensive laparotomy involves a great risk in acute pancreatitis, especially in its early stages. Many surgeons, and I must agree with them, have for this reason abandoned the operation for acute pancreatitis in the very first stage. But sometimes, when performing an exploratory laparotomy for peritonitis, we have to face a rather unexpected pancreatitis. In other cases we have to operate for gallbladder disease and find in addition an undiagnosed acute pancreatitis. When for certain reasons one is reluctant to perform a radical operation on these patients, it is apparently justified to perform a cholecystostomy for external drainage as the simplest and quickest procedure, provided the drainage of the bile through the cystic duct is secured. The best proof is the golden-brown color of freshly secreted bile. When the color of the bile is dark brown or greenish, it is a certain sign of stasis and obstruction of the cystic duct. There is obviously no use in performing a cholecystostomy for pancreatitis in such cases. Proper drainage can be achieved only when opening the common duct.

RADICAL OPERATIONS. These are the classical cholecystectomy and the electrosurgical operation. In all abdominal operations it is essential to secure an uneventful postoperative course. This can be obtained by covering all wounded parts in the operative field with serous tissue and before closing the abdominal wall to sew together any wound in the serosa caused by the operation. This procedure, which is called peritonization, has contributed a great deal toward lowering the operative risk. It should be most important to apply this sound principle when operating on the gallbladder as well. The difficulty lies in the covering of the liver bed, which frequently remains a wide bleeding area after the gallbladder has been removed in the classical way. Furthermore, small superficial bile ducts can be laid bare and bile may ooze out of this wounded area. In a state of acute inflammation the injured liver bed may be the source of a spreading infection and endanger the result of the operation. There is no doubt that the operative risk has proved to be much higher because of spreading infection and many other postoperative complications when operating during the acute stage rather than during the interval. The principle of the interval operation has been widely adopted. On the other hand, the surgical technic followed a different course and experiments were made to obtain peritonization in all cases of gallbladder surgery.

The so-called subserous cholecystectomy was further developed. The electrosurgical destruction of all infected tissues combined with the sewing of peritoneal flaps at last obtained the ideal conditions for peritonization. The mortality rate sank to a low level with this technic, 2 to 3 per cent, and there was no longer a greater risk when performing an early operation in the acute stage.

Having had experience with the electrosurgical operation since 1926 in more than 1,000 cases, I do not hesitate to say that this procedure certainly deserves preference over the classical cholecystectomy. The advantages of this operation are especially striking in urgent surgery when operating upon patients who are badly infected. It would give a wrong impression of my conviction, which is the result of long experience, if in describing both operations I were to leave the choice of the operation open to the student. Furthermore, the indications and favorable results mentioned previously are based also on the experience obtained with this procedure.

The following is a description of the technic which I now use as a routine:

Before the patient is placed in the correct position, the neutral electrode is bandaged on. The usual place is both thighs, but a rubber cushion covered by fine wire net on which the patient lies has also proved very useful. The patient is raised to a position of lordosis. In this position the liver is brought to the surface of the anterior abdominal

wall and the exposure of the lower surface of the liver is greatly facilitated. Furthermore, the pelvis is lowered, so that the intestinal loops fall into the lower parts of the abdomen and do not interfere with the operative field.

Incision. I have tried almost all the incisions described. Each has its drawbacks and advantages. But for fourteen years I have applied only the costal incision which undoubtedly has proved the most satisfactory in over 1,000 cases. The exposure is perfect and the healing tendency very favorable, so that I have not observed any incisional herniae or paralysis of the abdominal muscles even after long lasting drainage and suppuration. These experiences have been confirmed by Orator, Stegemann (in more than 700 cases), Mirizzi, and Usadel, who agree that the costal incision is the best for all operations upon the liver and biliary tract.

One of the favorable points of the costal incision in my opinion is that the abdominal cavity is opened very high and over the very spot of the operative field. This fact has proved to be important since we know that there is a certain local immunity in the abdominal cavity limited to the inflamed area. As long as we operate in this area, the tendency to heal is most favorable. We may even close the abdominal cavity without any drainage. The postoperative course is ordinarily uneventful, and movement of the bowels sets in after two days without any help. This need for not surpassing the immune area in gallbladder infections is not fulfilled when opening the abdominal cavity by the median, paramedian or transverse incisions underneath the costal arch.

Considering this fact of local immunity I also do not think it wise to perform a "by the way" appendicectomy through the same incision. When an appendicectomy is necessary, I think it more reasonable to perform the operation through a special small incision over McBurney's point. After having opened the abdomen and ligated all blood vessels thoroughly the round ligament is cut between two ligatures which are left long enough so that they can be tied together at the end of the operation. The falciform ligament is also incised and bleeding vessels, which are always found, are tied up.

Protection of the Abdominal Wall. Careful protection of the abdominal wall is important because it is sometimes impossible to avoid infected bile from soiling the operative field. This is hardly important in the immune intraperitoneal operative field, but it may interfere with primary healing of the abdominal muscles. For complete protection the following procedure has proved satisfactory:

The abdominal wall is covered first with gauze and then with oilsilk. This protection is held in position and fixed by special clamps during the whole time of the operation. A self-retaining retractor should always be used. It keeps the operative field open during the whole operation

without the help of the assistant's hands, which are not always steady and which may be used for other purposes.

Walling Off of the Abdominal Cavity. The operative field is walled off from the lower part of the abdomen and the left side by laparotomy pads moistened in warm saline solution. Each pad is secured by a tape and a ring. One roll of gauze is pushed downward under the abdominal wall; this holds back the colon and duodenum. The pad must be thick enough so that it is impacted between the posterior and anterior walls and is unshaken during the whole time of the operation. A second roll of gauze is forced between the vertebra and the anterior wall; this holds back the pyloric region. A third pad is introduced into the foramen of Winslow. In badly infected cases it is advisable to introduce a fourth pad between the convexity of the liver and the costal arch, thus protecting the subdiaphragmatic space.

When applying the costal incision it is never necessary to displace the liver. This should be avoided because traction on the diaphragm must be blamed sometimes for postoperative congestion and hypostasis on the right lower pulmonary lobe. The assistant holds up the liver gently with a spatulum when necessary.

The next procedure depends upon the particular findings. When adhesions are found around the bladder, they are divided with scissors and any bleeding should be thoroughly controlled. The adhering organs are the omentum, the colon and the duodenum. In these cases the walling off is performed only when the gallbladder has been freed. In the very acute stages of inflammation fibrin and gelatinous edema are frequently found. A separation in these cases can be done very gently with the finger. When the adhesions are firmer, we should make certain as to whether a localized perforation has taken place or whether a fistula between gallbladder and colon or gallbladder and duodenum has been established.

When the gallbladder has been freed from adhesions, it is advisable to empty it by means of a two-way syringe. The liquid content is aspirated and emptied into a receptacle (Fig. 138). The gallbladder is washed out several times with a warm saline solution and finally with a slightly antiseptic solution. The organ is then entirely emptied by aspiration and the needle removed from the collapsed bladder.

The whole process can be carried out without losing a drop of liquid. The emptying of the very enlarged bladder facilitates further dealing with the cystic duct which follows. The obstructing stone in the neck of the bladder or in the opening of the cystic duct is grasped between middle and forefingers of the left hand and the cystic duct thus straightened. The overlying serous covering is divided with long scissors and a silk thread is put under its proximal end by means of an aneurysm needle (Fig. 141). This ligature can be tied immediately, the thread

forming a good hold when the opening of the cystic into the common duct is laid bare. The operative field must be clearly visible before putting the distal ligature near the common duct. This maneuver of making the anatomy clear can be illustrated by the following sketches showing the different abnormalities. More frequently than one would imagine, the common duct is injured or even totally divided at this stage of the operation.

Fig. 105, a. This shows one of the most typical and normal aspects of the anatomy of the cystic duct. When pulling on the cystic duct, it is quite easy to place the distal ligature partly on the common duct, which then becomes strictured or even entirely ligated. It should always be a strict rule not to use clamps for the occlusion of the distal part of the cystic duct but to place a ligature which is tightened only when the anatomy is perfectly plain.

Fig. 105, d. This shows how the cystic duct winds behind the common duct and empties from the left instead of the right side. It is evident that in these cases it is very easy to grasp and ligate the common duct instead of the cystic duct.

Fig. 105, c. This sketch shows the cystic duct lying parallel with the common duct and emptying only over or into the papilla. This abnormality is especially important in the case of common duct stones. The surgeon tries to introduce a probe through the cystic duct, which must obviously be a failure in these cases.

A rare case of abnormality of the bile ducts is illustrated in figure 124 in which a single hepatic duct empties into the neck of the bladder and the bladder empties directly into the common duct. This abnormality can be described as a congenital absence of the cystic duct. Obviously in these cases cholecystectomy must lead to a defect in the single common duct, as I have experienced in one case. The picture shows how the immediate repair of the divided common duct had to be performed over a rubber tube (Fig. 126).

Summarizing, I should like to say that it is most important and practically always possible to deal with the cystic duct before removing the bladder or to perform electrocoagulation. The laying bare of the cystic duct is also important because no stones should be left in the cystic duct which may slip into the common duct, thus causing later complications and real recurrences in spite of the operation (Fig. 141).

I think the most important technical advice is that the use of a clamp instead of a primary proper ligature on the stump of the cystic duct adjacent to common duct should be strictly condemned. This use of a clamp is one of the main reasons for injury to the common duct.

Cystic Artery. It is always advisable to ligate the cystic artery when it is easily accessible and when not too much time is wasted in searching for it. The bleeding of the bladder is much lessened, otherwise we have to put ligatures on several branches which have to be divided later.

It is advisable, furthermore, to ligate the cystic artery in its entirety without dividing it, the reason being that when operating upon the common duct or also on the gallbladder, the central ligature may easily slip or be pushed away from the stump by some peritoneal pads or the spatulum of the assistant. In these cases a severe bleeding may occur. This may mean not only the holding up of the operation but it may

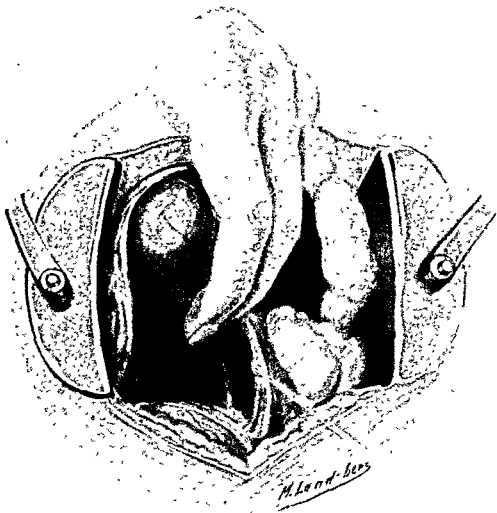


FIG. 141. Peritoneal covering of the cystic duct is divided and a ligature is placed on the duct.

sometimes prove rather difficult to put a clamp on the stump of the cystic artery which may slip under the common duct. When such an accident occurs during the operation, it is well to try to compress with two fingers the hepatoduodenal ligament and in it the hepatic artery before searching for the stump. But this accident can be avoided when the cystic artery is only ligated and not divided. There is no point in division.

Abnormalities of the cystic artery are very frequent. Usually it lies above and behind the cystic duct. But all sorts of variations are described which explain the fact that the artery sometimes cannot be found in its regular place.

The next step depends on the appearance of the serous layers of the

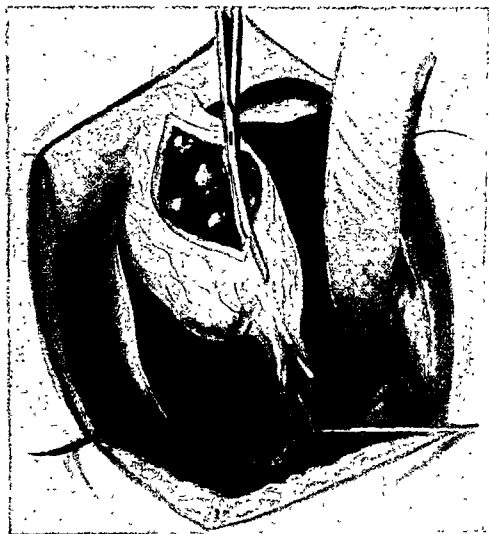


FIG. 142. Opening of the gallbladder.

bladder. When around the neck of the bladder the serosa is not too badly damaged, which is very frequently the case, it is divided and separated from the inner layers for the purpose of later covering this spot. Often a separation of the inner layers by saving the serosa can be carried out in the surroundings of the neck quite easily, even in badly inflamed cases. Partial subserous excision of the neck can be performed by saving very

valuable serous flaps. When this has been done the whole surrounding of the bladder is thoroughly packed again with gauze moistened with hot saline solution and the bladder opened with scissors from the fundus downward (Fig. 142). The stones are removed (Fig. 143). The part of the neck of the bladder, which has been separated from the serous layer is cut away; the edges of the divided bladder are grasped with several artery forceps and electrocoagulation performed (Fig. 144).

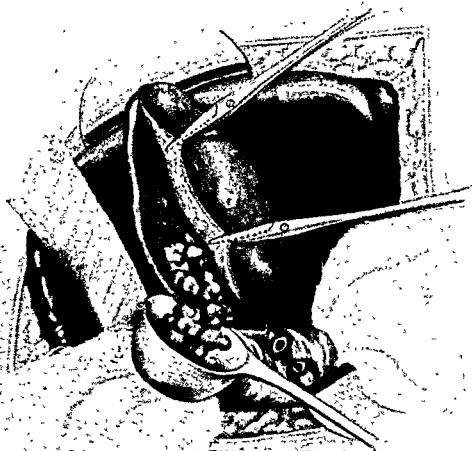


FIG. 143. Removal of the stones from the gallbladder, followed by excision of the gallbladder wall.

When the outer serous layer is healthy and inflammation restricted to the inner layers, coagulation should be limited to these inner layers, the outer layer being saved for the covering of the liver bed (typical mucoclasia) (Figs. 145, 146).

When the wall of the bladder is very thick, it is a good procedure to remove the coagulated scurf with a sharp spoon and then coagulate

again. By doing so, the depth of electrocoagulation can be better regulated. In some cases with a very thick wall I have removed the inner layers with an electric wire loop and then performed the typical electrocoagulation.

It is necessary to emphasize that all inner layers of the bladder should

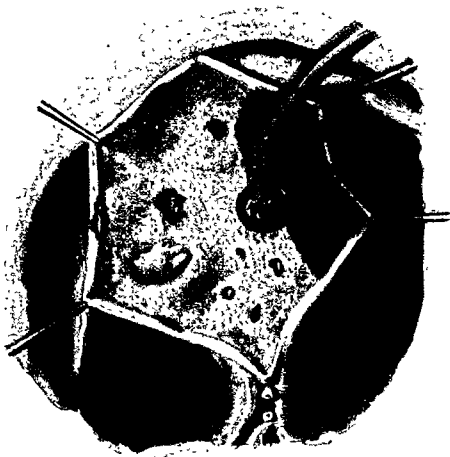


FIG. 144. Electrocoagulation (Mucoclasia) of the gallbladder wall adjacent to the liver. Serosa is preserved (Pribram's operation).

be destroyed by electrocoagulation and *only the serosa* saved. Very rarely are virulent germs found in the serosa of the bladder. It is a highly immune tissue with protective properties and with an excellent tendency to heal. It should be removed only when it is invaded by abscesses or when it is necrotic. Inflammatory edema of the outer layers is by no means an indication for removal. In case of a shrunk bladder, too, the outer serous layer can easily be preserved. The whole operation, with

opening of the bladder, electrocoagulation and the sewing up of the serous covering is very simple and can easily be performed in a few minutes. The contrast to cholecystectomy in these cases is very striking. The shrunken bladder has sometimes been removed like a malignant tumor, leaving a bleeding and badly injured liver bed. When the outer layer is badly damaged as well (which is especially true with purulent inflammation of the wall) the serous layer obviously cannot be preserved. Then the whole projecting part of the gallbladder is removed by electric knife or simple scissors at the level of the liver surface and only the remaining part thoroughly

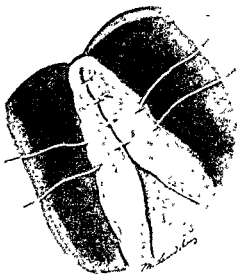


FIG. 145 Suture of the serous layers over the electrocoagulated liver bed (Pribram's operation).

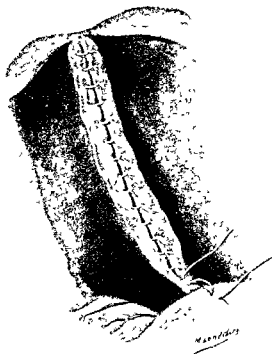


FIG. 146. Electrocoagulation of the gallbladder (Mucoclasia operation), final view (Pribram's operation).

electrocoagulated. Electrocoagulation of the liver bed should always be carried out carefully. Often there are abscesses containing stones hidden away at a depth of one-half to one inch. Electrocoagulation is, at the same time, the best treatment for any kind of liver abscesses. After electrocoagulation the operative field should be, and in most cases is, perfectly dry. When the outer layer can be preserved, the covering of the liver bed is very easily performed.

It is a widespread belief among most surgeons that ligation of the cystic duct is not safe and that in spite of the ligation leakage of

bile occurs in the majority of cases in the next few days following the operation, so that drainage is indispensable. I have always been puzzled by this. Why should a ligature in the cystic duct not hold firmly while a ligature around an artery of the same or even major caliber is perfectly safe in spite of the fact that this ligature not only is under considerable pressure but that the impulse of blood hammers in vain against this ligature seventy times a minute?

On the other hand, the pressure in the common duct is very low when there is no stasis and the outflow of bile does not run at all against the ligated cystic duct. I am perfectly satisfied that proper ligature around the cystic duct is as safe as the ligature around an artery. There must be other reasons for the leakage after ordinary cholecystectomy with drainage. These reasons are found entirely in the technic of removal of the gallbladder, leaving a wounded liver bed with open bile ducts.

The evidence for this statement is substantiated by the fact that when applying my technic neither after subserous excision nor after an electro-surgical operation has a leakage of bile from the cystic duct or the liver bed ever been observed in more than 1,000 cases.

After the gallbladder has been removed, the question arises as to what can be done with the electrocoagulated liver bed. When deep abscesses have been coagulated, I have found it best to leave a simple rubber drain the size of a small finger and not to bother about the covering. In other cases I have covered the liver bed with a free graft of omentum, which has proved the most suitable tissue for this purpose. To use a falciform ligament for this purpose was suggested by some surgeons, but I still believe that omentum is preferable.

The Question of Drainage. In most cases of electrocoagulation, especially when the inflammation is not too serious and a serous covering can be employed in a satisfactory way, no drainage at all is necessary and the abdominal wall can be closed. The postoperative course is practically always quite uneventful and satisfactory.

It must be emphasized (1) that when the cystic stump is ligated properly with silk and the cystic stump and liver bed are peritonized as already described, a leakage of bile never occurs. I had never regretted this primary closure of the abdominal wall under these conditions even in acute cases, empyema, etc. (2) When the whole gallbladder has to be removed by electrocoagulation, the question arises as to what is preferable: to cover the liver bed with a free graft tissue (omentum) or to leave a drain?

One should not be too rigid in sticking to any particular method. Every case should be treated individually according to the special conditions. A free graft in these cases serves a useful purpose only when primary closure of the abdominal wall is intended. It is, therefore, useless to cover the electrocoagulated parts when an additional operation on the common duct demands drainage. Furthermore, when deep abscesses,

especially after coli infection, must be coagulated, I prefer to leave the coagulated place as is and to allow drainage of the abdominal cavity through a simple rubber drain. In other and simpler cases, covering with a free graft tissue has had very satisfactory results. But even when allowing for drainage, it is always surprising how little secretion occurs from the coagulated spot.

There are cases in which gallbladder, colon and duodenum form one inflammatory mass, the size of a fist, in which dissection of the bladder and removal of the adhesions are extremely difficult and in which every attempt to do so causes hemorrhage. This is especially true when the gallbladder has perforated into the duodenum or the colon. The fibrous tissue around these fistulae can be felt as a firm thick ring. In these cases dissection of the gallbladder and opening of both fistulae should be avoided. The forced laying bare of the anatomy highly increases the operative risk. The healing tendency in these septic cases is generally very poor, evidently owing to the necrosis caused by *Bacillus coli*. As little as possible should be done in this badly infected field. An attempt to perform a routine operation often leads to a dangerous spreading of infection and increased absorption of toxic products, causing serious damage to the peripheral circulation. The bladder is often filled with fecal masses. Cholecystectomy and suturing of the opening in the colon after proper resection has a high death rate and entails many complications, such as postoperative colic fistulae.

A woman of forty-five, who had suffered for years from gallstone colic, had a very severe attack a fortnight before she was sent to the clinic in 1929. The agonizing pain ceased but chills and an evening temperature of about 102° F. persisted.

On admission she was a tall woman with flushed cheeks and slightly jaundiced conjunctivae. Leucocytosis was 12,000. In the right upper portion of the abdomen there was a painful lump as big as a fist. The diagnosis was empyema of the bladder or abscess after perforation. In view of the sepsis indicated by the swinging temperature, conservative treatment could not be continued.

The operation was performed under general anesthesia. A costal incision was made. The tumor was formed by the enlarged and freshly inflamed gallbladder, colon, duodenum and liver, all being covered with inflamed omentum. The slightest attempt to separate the organs was immediately followed by diffuse bleeding. The fibrous tissue forming the adhesions of the bladder with the colon was very dense and suggested perforation. By invaginating the finger into the colon, the site of perforation could be felt unmistakably. Separation of the colon from the bladder would have caused a large opening in the colon and was therefore abandoned. There could only be a question of either cholecystostomy, as an operation of emergency, or a radical mucoclasia. I decided on the latter.

Through a large needle yellow purulent masses with an offensive smell were aspirated. The gallbladder was opened as far away as possible from the site of perforation, and fecal semifluid masses, pus and stones were removed with a spoon. Stones were found down in the cystic duct. There was some difficulty in removing the impacted stone in the neck of the gallbladder. The liver bed was necrotic and full of abscesses. A curved electrode was introduced and the whole of the inside of the gallbladder was coagulated, all abscesses being carefully destroyed. The same was done with the whole inside of the opening into the colon. The neck of the gallbladder and the opening of the cystic duct were also carefully coagulated with a smaller electrode. The cavity of the bladder was filled with a piece of omentum, a rubber drain introduced near but outside the bladder and the abdominal cavity closed.

Apart from tachycardia, due to the thyrotoxic state of the patient, recovery was fairly satisfactory. The secretion from the drain was very small. The drain was gradually shortened and after a week's time was removed. The patient was followed up for many years. At first she had two recurrent attacks with colic. I am not sure whether these attacks were due to stones passing through the common duct which could not be examined at operation. After a year these attacks ceased and the patient was perfectly well, except for a certain intolerance to fatty food.

I do not think that any other method would have proved so useful in this serious case as mucoclasia.

Comments on the Electrosurgical Operation. The electrosurgical operation has reduced the operative risk considerably in all badly inflamed cases. The mortality risk is not higher than between 2 and 3 per cent. It allows a radical operation with a perfect healing tendency. It may be recommended as the operation of choice in urgent surgery.

Operation for a Ruptured Bladder with Biliary Peritonitis

(1) GENERAL PERITONITIS: (a) *Diagnosis Is Certain.* In these cases the procedure is exactly the same as when operating on an acute condition of the gallbladder. The abdomen is opened by costal incision and the bile aspirated by an electric aspirator. Meanwhile the gallbladder is covered with a saline swab so that the escape of stones into the abdominal cavity can be avoided. The self-retaining retractor is introduced and the operative field walled off from the rest of the abdominal cavity. The gallbladder is opened. The further procedure depends upon the condition in which the wall of the gallbladder is found. When it is badly damaged, it is cut away with the electric knife at the level of the liver and then thoroughly electrocoagulated; the cystic duct is ligated.

Now arises the question of drainage. The costal incision can in many cases be closed provided the operation was carried out properly with exact peritonization, as already described. This is especially the case in

a rupture due to hemorrhagic infarction. But if there is any doubt, it certainly is better to leave a drain at the lowest end (Fig. 147).

In case the bile has flown down to the lower parts of the abdomen, it is always wise to make a second incision in the median line or in the right part of the lower abdomen and to leave a drain there. A washout of the abdominal cavity is not necessary and not advisable.

The recovery and the postoperative course in these cases is favorable

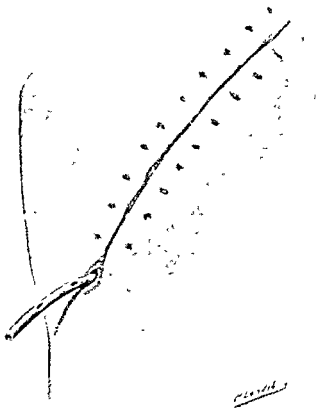


FIG. 147. Drainage of the abdomen through the lower end of costal incision.

when the time elapsed between the rupture and the operation has not been too long. Movement of the bowel starts sometimes only after the third or fourth day, probably due to temporary damage to the wall of the intestines through resorption of bile. The abdomen can be largely distended in these days but I have never had to perform a colostomy. With the reabsorption of bile the intestinal loops recover and the movement sets in. Of great value against this intoxication is a drip infusion administered during three or four days following the operation. Prostygmin or physostygmin one-twentieth to one-sixtieth of a grain may be added if necessary.

(b) *Diagnosis Is Uncertain.* In some cases we are not in a position to make any special diagnosis as to the origin of perforation, but must be content with the general diagnosis: acute condition of the abdomen with perforation. It may be difficult to distinguish between the perforation of a high or retrocecal appendix and that of a gallbladder. In these cases I think it advisable to make first a small incision over McBurney's point. The reason can be described as follows:

When operating over the appendiceal region no harm is done with this incision if the fluid proves to be bile, emptied through a perforated gallbladder, or gastric or duodenal content after perforation of an ulcer. On the contrary, the small appendiceal incision is very useful for the drainage of the lower portion of the abdomen in case of perforation.

But when we open the abdomen with a large incision over the gallbladder and the cause of perforation proves to be acute purulent appendicitis, we may, when exploring the abdominal cavity, very easily spread the localized purulent infection of the lower part of the abdomen into the upper parts, thus causing a general fatal peritonitis.

(2) **LOCALIZED PERITONITIS.** When through former attacks of inflammation extensive adhesions (pericholecystitis) have been formed between the bladder and the surrounding organs—duodenum, colon and omentum, or anterior abdominal wall, the contents of a ruptured bladder empty not into the free abdominal cavity but into the spaces between the adherent organs, forming a localized biliary abscess. Stones may also empty into this abscess which may spread downward along the ascending colon, causing more or less widespread infection in the abdominal cavity. In other cases the abscess may perforate into the subphrenic spaces, causing subphrenic abscesses, or it may even perforate into the abdominal wall and cause a cellulitis there.

It is most important in these cases strictly to observe the rule which has been described, i.e., to remain within the boundaries of the adhesions when operating. We can be quite certain that this purpose is achieved by opening the abdomen with a high costal incision. After having opened the peritoneum, a bile-stained pus immediately pours out in some cases. We should not be afraid of this event; it looks more serious than it actually is. It is certainly not necessary to finish the operation at this stage by putting in a drain. We can perform a radical electrosurgical operation in these cases, too, thus diminishing rather than increasing the risk of the patient's condition.

The following case is an illustration of the procedure:

A sixty-six year old woman had to be operated upon as an emergency after a serious attack of colicky pain in the right upper part of the abdomen with peritoneal irritation spreading and rapidly increasing. She was slightly jaundiced and vomited. Her temperature was about 100° F. She had had several previous attacks of colicky pain but never as severe

as the last one. The diagnosis of perforated gallbladder was likely.

The operation was performed under spinal anesthesia and gas. The peritoneal cavity was opened and immediately some purulent liquid appeared which was aspirated. Everywhere there were signs of fresh inflammatory adhesions between colon, omentum and duodenum, The wall of the bladder was very thin, black-stained and necrotic. A number of gallstones were found in the abdomen. Some more pus was aspirated between the adhesions. The necrotic part of the gallbladder was cut away and stones removed. The liver bed was thoroughly electrocoagulated. The cystic duct was divided between two ligatures. Further inspection showed an abscess in the liver bed the size of a small apple containing pus and stones. The content of this abscess was emptied by aspiration, the stones removed and the abscess electrocoagulated. The operative field was now dry and clean. The common duct was palpated; the wall was thin and no stones could be felt. A piece of corrugated rubber and a drain were left, and the abdominal wall was closed. Recovery was uneventful and satisfactory. Very little drainage took place and on the tenth day all drains could be removed.

To summarize I must point out that when performing a simple drainage or even a cholecystectomy, the abscess in the liver bed would certainly be overlooked. Nor would the safe examination of the common duct be possible without the antiseptic measure of electrocoagulation.

BILIARY PERITONITIS WITHOUT PERFORATION. There is a number of such cases referred to in the literature and I myself have been able to observe a few since. Nevertheless, a certain degree of skepticism and a careful search for a small or concealed perforation are advisable before making this diagnosis. In one of my cases a very small perforation in the common duct could be detected as the source of biliary peritonitis.

In another case I found the whole tissue surrounding the cystic duct and all of the hepatoduodenal ligament infiltrated with bile, while the wall of the bladder itself looked normal. I could not find any perforation neither in the gallbladder, nor in the common duct or cystic duct. But I had the impression that the bile was penetrating through the wall of the common duct rather than the wall of the bladder, the reason being the reflux of pancreatic juice which increases the permeability of the wall. I found this confirmed when observing a case of biliary peritonitis without perforation but with an acute pancreatitis.

Treatment. These patients are operated upon as emergencies under the signs of general peritonitis. When finding biliary peritonitis without a ruptured bladder, we should be content not only to remove the bladder, but having in mind the reflux of activated pancreatic juice into the bile ducts, we should always open the common duct and introduce the proper external drainage.

ACUTE OBSTRUCTION OF THE COMMON DUCT IN THE CASE OF INFECTION (CHOLANGITIS)

Acute obstruction of the common duct is a matter of urgent surgery in all cases of infection of the bile ducts. There seems to be no more difference of opinion about this. Infection may flare up in a virulent way. The former latent cholangitis spreads around the small bile ducts and capillaries while multiple abscesses develop. Septicemia may result. Furthermore, the stasis of bile in these badly infected cases leads very soon to break down of liver function (cholemia). But even in noninfected cases the danger arising from any delay in removing the obstacle has been evidently proved.

The opinion that in jaundiced cases one should wait for several weeks before deciding to operate is certainly not justified. This point of view, held until recently by many physicians and even surgeons, has been responsible to a large extent for the high operative mortality.

Obstruction of the papilla is caused in the majority of cases by a stone which has slipped from the bladder through the cystic duct into the common duct. The obstruction becomes complete when a muscular spasm sets in around the stone. Experience has shown, in contrast to previous opinion, that a severe colicky pain is caused mainly by the stone passing through the cystic duct, while the passing of a stone through the papilla is sometimes painless. Even a long lasting spasm in the papilla may be entirely painless. This seems to be important because it is generally believed that a colicky pain in these cases means that a stone passes through the papilla, which is not true.

Chills are observed in practically all cases of infection; jaundice increases quickly; the feverish redness of the cheeks combined with a yellowish color gives the patient the characteristic septic-cholemic appearance. Fatty degeneration of the liver, which sets in very rapidly, is in some cases quickly followed by acute insufficiency and coma.

The post mortem examination shows a difference between the acute and the subacute or more chronic cases. In the first instance the liver is rather small, flabby and in a state of extreme fatty degeneration. In the subacute cases the liver is sometimes of a hard consistency because of an increase of fibrous tissue, but the fatty degeneration is practically always pronounced. The glycogen content of the liver cells is small, and sometimes glycogen is practically absent.

TREATMENT. The aim of the operation is to remove the obstruction and to secure a free outflow for the infected bile from the common duct. Since the operation is an urgent one, we cannot waste too much time on long preoperative measures. Nevertheless, a certain course of preoperative treatment is advisable for a few days if the case permits it. Every case should be handled according to its particular findings. Our pre-

operative treatment consists (1) of an intravenous injection of 40 cc. of a 50 per cent solution of glucose with a 10 per cent solution of calcium gluconate; (2) intramuscular injection of 5 units of insulin; (3) as a prophylactic measure against cholemic hemorrhage, we give parathormone, 0.5 cc. to increase or maintain the calcium level of the blood; (4) for the same purpose an oily solution of vitamin K in capsules with dehydrocholic acid or ox-bile is administered; and (5) local application of an icebag or cold fomentation on the whole region of the liver.

This preoperative treatment is carried out for two or three days. Then we are practically always in a position to consider whether or not there is a chance left for spontaneous improvement. When there exists a definite tendency of the bilirubin content of the blood serum to decrease, we can assume that spontaneous drainage of bile into the duodenum is improving, i.e., that the stone or the stones which are the immediate cause of the acute obstruction have passed into the duodenum or that the spasm around the stones has relaxed allowing a certain outflow of bile.

In these cases and only in these cases are we entitled to wait, because not only the general state of the patient is likely to improve but the tissues also may be cleared of the bile dyes so that all conditions for the operation become more favorable. The liver function improves and the danger of postoperative cholemic hemorrhage diminishes.

But a postponement of the operation beyond the time when the bilirubin level in the blood has again become normal serves no useful purpose except when the special condition of the patient calls for further treatment or delay.

We must realize that vanishing of the jaundice is by no means proof that the common duct has become free of stones. On the contrary, even after a so-called "successful attack" with total disappearance of jaundice we find the common duct almost always still filled with stones. Therefore, we must take advantage of this jaundice-free interval because we are never sure whether a new attack with a new obstruction will not take place in the very next few days.

When, on the other hand, the bilirubin in the blood shows no tendency to decline following the attack but rather to increase or come to a standstill, we should perform the operation without delay even when the course is not complicated by high temperature and chills.

There is no use in waiting for a spontaneous passage of the stone, since the chances are very small and the danger caused by the delay extremely high. Each day that the operation is postponed may cause irreparable damage to the liver or may give rise to a fatal septicemia. In some cases an acute pancreatitis may be the last fatal complication. The liver may soon be damaged to such a degree that an acute hepatic insufficiency may set in a week after an otherwise successful operation.

Improvement in the surgical technic and the simplification of the operation in these cases, implying a greatly diminished operative risk, have justified this point of view in the last decade.

In order to carry out the treatment properly and in the most reasonable way we should bear in mind the following danger points for complications: (1) hemorrhage; (2) the development of general septicemia, with multiple abscesses around the small bile ducts and also in other organs; and (3) cholemia and acute hepatic insufficiency.

ANESTHESIA. I strongly advocate the use of spinal anesthesia in all these cases with an additional small dose of gas-oxygen. The addition of gas-oxygen is useful to secure proper ventilation and regular breathing during the operation. We must realize that all general anesthesia even ether is toxic to the liver cells, as has undoubtedly been proved by experiments.

There is a definite contraindication to the use of any products such as evipan, amytal, avertin, pernocton and all drugs of these chemical groups. After using such anesthetics I have observed that patients are sometimes not able to recover from a drowsiness which has gradually led to fatal hepatic coma within eight or ten days.

Nor do I advocate local anesthesia in these cholemic cases, since I have sometimes observed abundant hemorrhage at the points of injection. Besides, we need complete relaxation of the abdominal wall, which cannot be achieved to the same extent by any other anesthesia than a spinal one. We use percain, and inject between the second and third lumbar vertebrae.

The procedure at the beginning of the operation is the same as that which I described when discussing the electrosurgical operation. When the peritoneum is opened the left hand explores provisionally the pathology of the special case enabling us to make a proper plan for operation. In the majority of cases of common duct stones we find the gallbladder medium sized or even small and shrunken. We satisfy ourselves regarding the condition of the cystic duct and feel the common duct downward to the papilla.

I always operate on the gallbladder first before opening the common duct. I think this procedure offers several advantages although it is not generally adopted. The advantages are that the exposure of the common duct is simpler and the operative field wider. Secondly, an infective spot is removed and after electrocoagulation the liver bed is transformed into a dry, practically sterile place.

In cases of jaundice it is always wise to ligate the cystic artery as a routine in order to diminish the bleeding. The gallbladder is opened and electrocoagulated. I practically always limit electrocoagulation of these cases to the inner coats, preserving the outer layers in order to cover the

liver bed carefully. When this has been done properly, we deal with the common duct.

Some surgeons advocate the exploration of the common duct by splitting the cystic duct downward. There are many objections to this procedure which I personally perform only in rare and exceptionally suitable cases. The main objection is that the cystic duct shows anatomical abnormalities frequently. The opening in the common duct is sometimes very low down so that it runs parallel to the common duct. Therefore, we explore only the cystic duct and not the common duct.

The cystic duct opens frequently into the common duct in a pointed angle so that it is very difficult to introduce a probe upward into the hepatic duct, a procedure which is very important for the exploration of the hepatic duct for stones. The hepatic duct or rather both hepatic ducts should be investigated by introducing a probe as high as possible into the liver. I could in a number of cases discover and reach some stones in this way which I certainly would have overlooked by omitting this procedure. Therefore, I believe the exploration of the common duct through the cystic duct is not a safe and satisfactory procedure which could be recommended.

The hepatoduodenal ligament is divided downward with scissors to expose the common duct in its whole length. Any blunt dissection is to be avoided in order to preserve the flaps for the eventual peritonization of the common duct which has been laid bare.

Bile from the common duct is aspirated with a fine needle. This procedure is always useful not only to empty the common duct before opening and to check the slipping of stones but also to assure ourselves that it is really the common duct we are about to open. In some cases with difficult anatomy, especially when performing a "secondary operation" for recurrent stones, this may appear very valuable. After aspiration the common duct sometimes represents a compact chain of stones or a solitary stone appears clearly in its contour.

The common duct is opened best low down in the supraduodenal region about one-half to one inch over the border of the duodenum. If possible, the incision should avoid the small veins generally lying in the anterior wall. When severed they should be ligated. Sometimes the blood control can be secured with a swab of cotton wool moistened with a 60 per cent glucose solution.

PROCEDURE WHEN A SOLITARY OCCLUDING STONE IS PRESENT. These cases are very easy to deal with. The gallbladder is practically always shrunken. The cystic duct is usually wide and the common duct enlarged. It is easy to push the stone, which may be the size of a hazelnut or even a chestnut, upward into a suitable position. The stone is held with two fingers of the left hand; the common duct is incised over the

stone, preferably in a transverse direction, and the stone is removed.

In most cases it is easy to pass a probe through the papilla into the duodenum. Gentle dilatation by increasing the size of the probes can be carried out without difficulty. No duct drainage is necessary provided the bile is not too badly infected.

We may consider the use of a primary suture of the common duct. In case of a badly damaged liver showing a flabby appearance after decompression I am inclined to favor primary suture in order to avoid the serious complication of acute insufficiency after decompression. This sometimes appears even in the later course between the fifth and eighth postoperative days. The maintaining of a certain amount of intraductal back pressure is important in these cases. We shall deal fully with these post-operative complications later on.

Before using the method of a controlled back pressure and cholangiogram as a routine, I frequently used a primary suture of the common duct, especially in cases of a badly damaged liver, in order to avoid the serious complication of acute insufficiency after abundant external drainage without back pressure. When draining the normal way into the duodenum, a certain amount of back pressure is maintained even when there is abundant outflow of bile.

Technic of Primary Suture. The condition for a suture of the common duct is of course that we are perfectly satisfied that the papilla is free. But we should also prevent postoperative spasm. Therefore, dilatation of the papilla should always be performed where a suture is intended.

I prefer fine silk kept in sterilized liquid paraffin oil, as used for the sutures of blood vessels, for the suture of the common duct. Even the finest catgut is not suitable for this purpose and not safe enough because during the absorption the catgut swells, and the holes in the wall becoming wider leakage may occur after a few days.

The first layer alone of suture of the wall is rarely tight enough so a second layer should be placed. This is done by sewing the hepato-duodenal ligament on this first row with a few stitches for which we may use a normal fine silk. When doing so the suture becomes absolutely safe so that one can perform a primary closure of the abdominal wall without any signs of post-operative leakage.

The advantage of such a primary suture is that it diminishes considerably the danger of cholemic hemorrhage in the presence of long lasting jaundice. The main thing is that the abdominal wall should not be closed until all oozing points have been controlled and the operative field appears perfectly dry. But when a surgeon does not feel quite safe about his suture, he should leave a drain for safety. However, he will observe that in spite of the drainage no leakage of bile takes place when the suture has been properly placed.

PROCEDURE WHEN MULTIPLE STONES ARE FOUND. The common duct

is incised in the supraduodenal region longitudinally or transversely when the common duct is sufficiently wide. The presenting stones are removed with a spoon. The left hand grasps the hepatic duct and manipulates the stones downward. When the hepatic duct has been emptied,

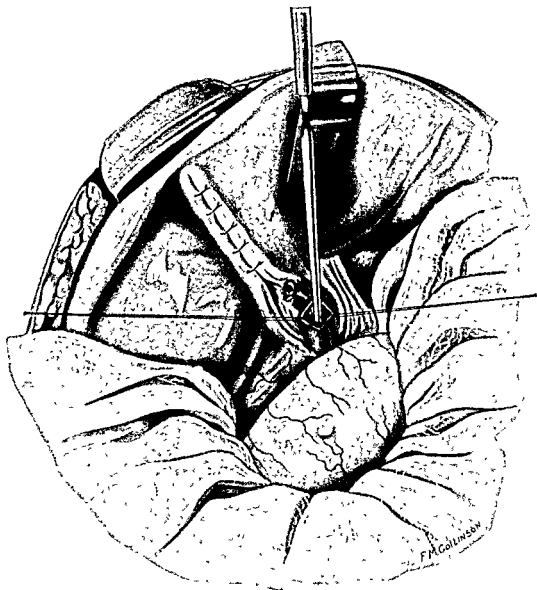


FIG. 148. Probing of the common bile duct for stones. The blunt end of the probe is palpated through the anterior wall of the duodenum.

the stones from the common duct are worked from the papilla toward the incision, but care should be taken so that no stones slip upward again into the hepatic duct. Stones over the papilla may sometimes be so tightly impacted that we need a forceps or a sharp spoon to remove them.

When we come to the point at which no stones can be removed, we have to make certain that the papilla is free from stones. We try to introduce a probe of small caliber gently through the papilla into the duodenum (Fig. 148). It is sometimes not easy to say whether the probe has really passed into the duodenum. It is a good practice to grasp the button of the probe through the anterior duodenal wall. Thus we can easily ascertain that the probe lies freely movable in the duodenum. Another proof whether the papilla is free is the following:

Through a rubber tube which is held tightly in the common duct between two fingers we inject saline solution. If no saline solution flows

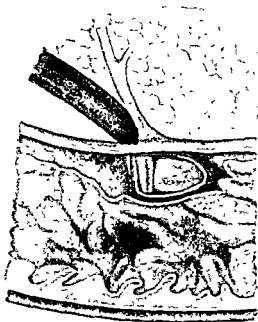


FIG. 149. Passing a probe over a stone in the dilated papilla. No force should be used on account of possible perforation.

back after removing the syringe we have proof that the liquid has passed; we may also inject a few cubic centimeters of hydrogen peroxide into the common duct. When this passes through the papilla, the duodenum becomes distended and ballooned by oxygen bubbles.

However, this proves only that the papilla is to a certain degree permeable, not that all stones are removed. Even stones the size of a cherry pit lying in the dilated suprapapillary region may allow the probe to pass freely into the duodenum and palpation of this suprapapillary region around the probe may not give conclusive evidence, since it is frequently surrounded by dense pancreatic tissue. We must face the fact that for these reasons one can never be certain that he has not over-

looked stones that may give rise to recurrent troubles, thus nullifying the beneficial effects of the operation.

On the other hand, there may be hard and calcified lymphatic glands around the common duct which are assumed to be stones. There is a great temptation to grasp these hard glands between forefinger and thumb of the left hand and to try to remove them with a forceps through the supraduodenal incision. Obviously, this attempt to grasp the supposed stone fails or the maneuver leads to perforation with heavy bleeding when jaundice is present. The best and at the same time most harmless means of detection is an x-ray examination (cholangiogram), a procedure which must be considered a great advancement during recent years and which should in these cases always be used as a routine.

The passing of probes through the papilla must be carried out with the utmost care and no force should be applied. Perforation of the common duct in the lower retroduodenal region may easily occur and is more frequent than might be expected (Fig. 149). It may be an accident with a fatal outcome. We must remember that here there are small vessels as well as the pancreatic tissue frequently surrounding this region which may easily be damaged. Here any injury may give rise to cholemic bleeding in the days following operation or leakage of pancreatic juice followed by local peritonitis and serious toxic symptoms. Furthermore, the tissue of the supraduodenal region has not the same immunity against infection and a spreading cellulitis because of infected bile leaking through the perforation may take place behind the duodenum.

Forcible Dilatation of the Papilla. Many surgeons advocate this maneuver and insist that it be performed in all cases of stones of the common duct. In many instances this dilatation can be done easily by increasing the size of the probes. The procedure is sometimes very useful and there is no doubt that it can be adopted without doing any harm if performed gently and with the greatest care.

But there are strong objections to forcible dilatation. The danger of making a false passage and perforation have already been mentioned. Furthermore, the danger of provoking hemorrhage, especially in the presence of prolonged jaundice, is considerable.

Recently published research work has shown that the result of such dilatation is not only transitory, passing after a short time, but may even cause a cicatricial shrinking of the papilla. In a number of cases the obstruction of the papilla is not wholly or even partly due to stones, but due to a spasm of the sphincter which may last during the whole time we are attempting to pass through the papilla.

An attempt to overcome this spasm by forcible introduction of the probe must be condemned. I am perfectly convinced that one of the reasons for the great operative risk and high mortality in cases of ob-

structive jaundice is the forcible attempt to overcome resistance in the papilla. As a matter of fact, mortality in my own statistical figures has dropped considerably since the development of a different, more gentle and safer method of dealing with impacted stones in the papilla (ether method). Whatever method we use we should follow the rule that when all stones cannot be removed easily from the supraduodenal incision, all forceful attempts should be stopped and the operation continued by other means.

CHOLANGIOGRAPHY DURING OPERATION. Mirizzi has advocated performing a cholangiography on the operating table in doubtful cases. The procedure is as follows:

Ten to 20 cc. of the opaque substance (lipiodol) are injected through the bladder, or if the latter has already been removed, through the cystic duct, or directly injected into the common duct before the distal ligature has been made or after the primary ligature has been removed.

Of course, there must be an operating table fitted for this x-ray examination. In many instances this procedure has been very helpful in clarifying the condition of the common duct as to whether or not there were obstructing stones. Also spasms of the sphincter of Oddi and the lower part of the common duct may be detected.

However, there are some objections which should be mentioned:

(1) The x-ray examination prolongs the operation. This fact is especially important in poor risk cases with obstructive jaundice and septicemia.

(2) A single picture may give no clear evidence and may even be misleading.

(3) A spasm in the papilla, which is quite frequent and may last for a long time, may be mistaken for a mechanical obstruction.

(4) Not many hospitals are in the fortunate position of having an x-ray apparatus in the operating room. Therefore, I think this x-ray examination on the operating table will hardly become a routine procedure in dealing with common duct stones.

(5) Finally, the introduction of the ether method, which we will discuss later, has shown that we are able to free an obstructed papilla even in the postoperative course.

Drainage. External drainage of bile is essential in controlling in the quickest and most successful way acute infection of the bile ducts (cholangitis). It should always be applied in cases with complicating pancreatitis. The effect of external drainage is an immediate and dramatic one. The temperature becomes normal; the chills stop and the bile formerly purulent and turbid clears within a few days to a normal, yellowish-brown color. Indeed, the value of this external drainage has been appreciated by all surgeons and it has been applied widely in the last fifty years since the popularization of gallstone surgery.

But it seems necessary to point out that external drainage may involve some definite dangers, and the thorough understanding of these dangers has influenced the indications and changed our technic.

When we remove the obstructing stone, the pressure in the bile duct immediately drops to zero and even below, especially when suction drainage into a bottle is performed. This acute decompression and the absence of any pressure in the bile duct, when such external drainage is used, may cause serious damage to the liver and lead to a complete breakdown of its function.

We may distinguish between the damage caused by acute decompression, when removing the obstructing stone, and the damage caused by the absence of bile in the intestines in the presence of long lasting drainage. The clinical facts are best illustrated by an example:

We operate upon a patient with obstruction of the common duct that has lasted for weeks and even months. Jaundice is considerable but the patient apparently is still in a satisfactory state of health. The liver works just enough to meet the daily demands. The operation is quite easy and can be performed quickly. The ampulla of Vater can be easily freed by a supraduodenal incision. We introduce a drain into the common duct and the prognosis seems to be quite favorable. The first three or four days following the operation are satisfactory and recovery appears normal. Intestinal motion has already set in and the patient seems to be saved. From the fifth to the eighth day a tendency toward sleep causes some worry as the first sign of a turn for the worse. The drowsiness increases during the next few days; the patient sleeps almost the whole time and can be kept awake only with difficulty and only for short periods. Between the eighth and tenth day following the operation the patient dies.

One feels that before the operation there was still a certain functional equilibrium of the liver in spite of the fact that the liver cells were extremely damaged. It appears that the removal of an obstacle, although obviously noxious, has caused a total collapse of liver function. I have called this state "acute insufficiency of the liver following decompression." It seems that a sudden decrease of pressure in the common duct, after opening it and removing the obstruction, must be regarded as one cause at least of collapse of the organ.

We must distinguish two forms of such an acute functional collapse after decompression. In one form the state of the heart and circulation is sufficient until the last; in other cases cardiovascular symptoms following such a sudden change in the bile duct prevail from the outset. Both types, however, end in a coma which must be regarded as the immediate cause of death.

I have tried to find an explanation for this acute cardiovascular collapse. The liver is not only an admirably working chemical factory, but

also an organ influencing the blood circulation. The liver is able to store 20 per cent of the total amount of blood, and because of this ability is a direct circulatory assistant to the right side of the heart. It relieves the right auricle in the case of insufficiency. An obstacle in the common duct causes increased pressure and dilatation of the intrahepatic bile ducts. A sudden decrease of this pressure to zero or even below causes an afflux of blood, an increase of the blood store in the liver and the diminution of the amount of circulating blood. It may thus give rise to a serious collapse of the peripheral circulation, especially when this has already been damaged. This collapse following the removal of an obstacle and a sudden fall of pressure is well known in the case of tapping of an ascites, a pleural effusion or even after an abrupt emptying of a full bladder by catheterization. This organ collapse and the acute insufficiency of the liver, following removal of an obstruction and sudden decompression, has been the subject of study for several years.

In other cases the cardiovascular symptoms are not so pronounced, the pulse can be regular and is rather slow; clinically, the acute hepatic insufficiency and intoxication leading to coma are the outstanding features. At the postmortem examination the liver is found to be small, flabby and in a state of extreme fatty degeneration. Microscopically, we find hemorrhages in the parenchyma.

Another damage is a more chronic one and is observed only in long lasting external fistulae; but it occurs mainly in such cases in which practically no bile at all finds its normal way into the duodenum.

This damage must be considered as a sequel to the deficiency of bile. The following mechanism seems to be the most likely: Bile, and especially bile-acids are necessary for the proper depletion and absorption of food, especially fats. When bile is absent, important fat-soluble vitamins, as vitamins K and D, are not absorbed, which results in a deficiency of these vitamins. The pathological changes observed are anemia, general fatigue, debility and atrophy of organs, especially osteoporosis.

These changes brought about by external drainage must be faced when considering indications and technic. The rules to be observed when performing external drainage are: (a) The fall of intraductal pressure must be avoided and back pressure artificially maintained. (b) The bile drainage into the intestine must be secured as quickly as possible and, as a rule, not extended over six to eight weeks.

Technic. I have given up entirely the drainage with a T-tube which has been widely used. I introduce only a simple rubber tube with a side hole downward to the papilla and allow drainage only as an overflow (Fig. 150). Direct drainage of the hepatic duct is never performed. This rubber tube is sewn water-tight into the incision, the water-tightness being secured by stitching the hepatoduodenal ligament around the tube. This water-tight condition is essential for maintaining back pres-

sure. The size of the tube is chosen so that the bile draining from the liver can easily pass alongside the tube into the duodenum. When the operative field is perfectly dry and covered with peritoneum, as it always should be before closing the abdominal wall, this rubber tube is allowed to pass into the lower angle of the abdominal incision. When the operation has been carefully performed, it is not necessary that another tube to drain the abdominal cavity be introduced, but no harm is done when another drain the size of a small finger or a small corrugated rubber is

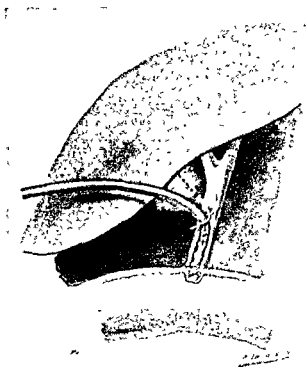


FIG. 150. Drainage of the common bile duct.

left for safety. This second tube is removed as a rule after forty-eight hours when no leakage alongside the drain occurs. To maintain the back pressure the rubber tube is closed by a clamp immediately after the operation and left there for several hours. The clamps can be released for a few minutes to allow an outflow and then should be closed again. No continuous drainage of bile should be allowed during the first twenty-four hours after operation. This method has proved quite satisfactory for eight years.

Another method to maintain back pressure and to regulate it during drainage is as follows:

The drainage tube is first connected by a U-shaped or Y-shaped glass tube which is fixed to a wooden board. This board can be elevated or lowered on a stand. We start usually at about 25 cm. above the level of the common duct. The drainage of bile can be properly regulated by

maintaining the pressure at the desired level. This procedure is also of definite value because it forces a certain amount of bile through the papilla into the duodenum, thus avoiding an unnecessary loss of bile. The possibility of a serious toxic state following acute decompression has been recognized by many authors, such as Mont H. Reid, when advocating cystocholecholestomy, also Ravdin, Frazer, etc.

I think this method of drainage should be generally adopted and should replace the routine method formerly used which allowed an unlimited outflow into the drain and the dressing.

But I also wish to emphasize that for the purpose of maintaining pressure the water-tightness of the tube sewed into the common duct is essential. The sequels to a chronic loss of bile can be avoided by maintaining a certain pressure in the common duct just mentioned, so that right from the beginning a certain amount of bile flows into the duodenum.

Chronic bile fistulae, sometimes lasting for months, have been observed frequently. They are practically all cases of a stone which has been overlooked in the common duct. One can understand that surgeons have been very reluctant to suggest a second operation and have preferred taking the chance of allowing the stone to pass into the papilla spontaneously. As a result these patients lost bile for a long time followed by general debility, anemia and osteoporosis. When the patients finally had to be reoperated upon, the operative risk was obviously high. By means of the ether method under x-ray control this mishap can be avoided with certainty. I have not observed any bile fistula in my own patients during the last decade.

OPERATIONS FOR OBSTRUCTED STONES IN THE PAPILLA. When our attempts to free the papilla by the supraduodenal way have failed and there are stones left in the ampulla which cannot be removed, the following operative procedure can be considered:

Retroduodenal Method of Laying Bare the Papilla. With a soft clamp the blades of which are covered with rubber, the common duct is temporarily closed so as to avoid further leakage of bile. The duodenum in its descendent part is grasped with the fingers or pushed with swabs to the left side. The posterior layer of parietal peritoneum on the right border of the duodenum is incised. In jaundiced cases it is always wise to divide this peritoneum between several ligatures because this part is especially inclined to bleed, being a dangerous spot for postoperative cholemic hemorrhage. Therefore, any bleeding must be carefully controlled here.

Then the duodenum is turned to the left side and in this way the retroduodenal part of the common duct is exposed (Fig. 151). As has already been mentioned, this part is richly vascular and frequently

surrounded by pancreatic tissue. It is always wise before incising at the stone to try to push the impacted stone upward. We may succeed in some cases, thus avoiding the need for an incision. When this maneuver fails, we try to needle down the stone with a fine needle. What we feel

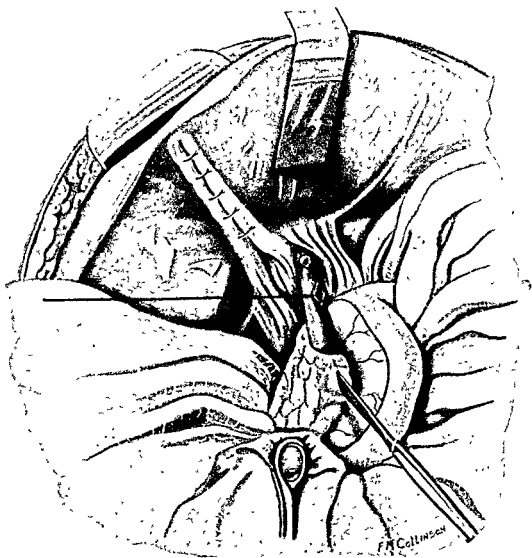


FIG. 151. Retroduodenal papillotomy.

may only be a gland and we avoid in this way a very disappointing and even dangerous incision.

We may also succeed in loosening the stone by this maneuver and then be able to push it upward into the common duct. We may feel the stone quite easily as the incision is made right over the stone and remove

it without difficulties. The diathermy knife is preferable to the ordinary one for this purpose. The bleeding, which may be considerable, should be controlled very thoroughly, but it may prove rather difficult in this pancreatic tissue. Through the incision we pass probes downward to the papilla and satisfy ourselves that it is now completely freed.

After having freed the papilla from stones the incision is closed with a few fine silk sutures. A common duct drainage as described should always be performed through the supraduodenal incision, and a safety drain should be placed in the abdominal cavity as well.

Comments. The mobilization of the duodenum to explore the retroduodenal part of the common duct is useful in cases in which we find some difficulty in passing a probe through the papilla from the supraduodenal incision. We may guide it easier into the duodenum and may even, as has been mentioned, work up some stones from this part toward the incision. We may also determine whether the cause of obstruction is really a stone, an indurated pancreatic tissue or gland, or only a spasm. But there are serious objections to the transpancreatic opening of this part of the common duct.

POSTOPERATIVE HEMORRHAGE. This danger is especially high in cases with long lasting jaundice. This retroduodenal part of the common duct is surrounded by small arteries and veins—branches of the pancreatic or duodenal vessels—which must be divided when opening the common bile duct. The bleeding from the pancreatic tissue is sometimes difficult to control and a postoperative hemorrhage may easily occur from very small vessels which do not bleed at all during the operation.

Furthermore, any injury to the pancreatic tissue endangers considerably the tendency to heal because of the leakage of pancreatic juice, thus increasing the operative risk to a large extent.

There is also the danger of spreading infection in the loose retroduodenal space, the immunity and resistance against infection here being much less pronounced than in the intraperitoneal cavity. I was sometimes deeply impressed at the postmortem examination of such cases to see how in those seriously infected patients the infection had spread without any considerable healing reaction, while in the intraperitoneal part the infection had already been controlled and brought to a standstill. This retroperitoneal cellulitis must be blamed at times for the fatal outcome in such cases. Therefore, I have given up this retroduodenal choledochotomy altogether.

THE TRANSDUODENAL CHOLEDOCHOTOMY. This operation in its technic and idea is clear and not too difficult. When the common duct is obstructed and the stones apparently are not easy to remove from the papilla, we can decide at the very beginning not to open the common duct in the supraduodenal part but to lay bare the papilla right through

by opening the duodenum. However, in most instances we have already opened the common duct by a supraduodenal incision but have been unable to remove all the stones, therefore, we decide to lay bare the papilla by the transduodenal way as an additional operation.

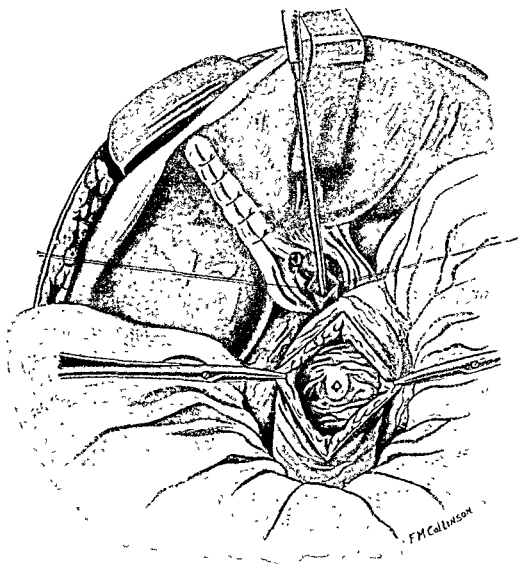


FIG. 152. Retroduodenal and transduodenal choledochotomy.

Technic. The peritoneum on the right border of the duodenum is incised between ligatures. One finger goes behind the posterior wall, mobilizing the whole middle of the second part of the duodenum. But this is not enough. The anterior wall, too, must be laid bare and freed from adhesions or congenital membranes, which are frequently found con-

necting this region with the colon. One feels the position of the papilla and opens the duodenum by a transverse or longitudinal incision (Fig. 152).

It is well known that in most cases the duodenum is opened too high

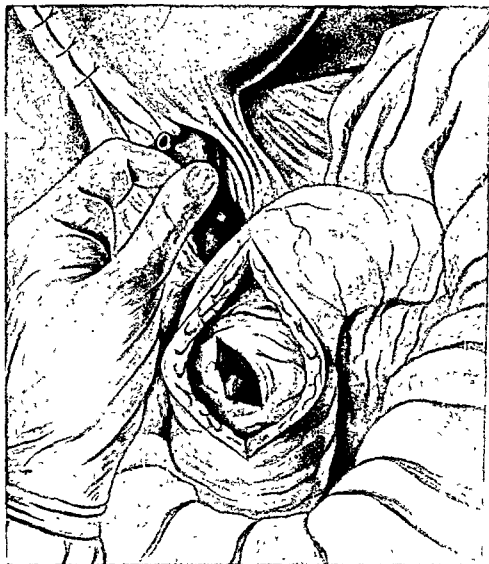


FIG. 153. Transduodenal choledochotomy.

when the obstructed stone in the papilla is our guide. Therefore, it is always advisable to make the incision half an inch or even an inch lower than we would expect the papilla to be. A swab secured by a thread is pushed upward toward the pylorus and another one downward to check the passage of the duodenal content. When the incision in the duodenum has been made in the right place, it is not difficult to locate the papilla

and feel the impacted stone. In most cases the position of the stone does not correspond exactly to the position of the papilla. It is not necessary to make our incision right on the papilla in order to remove the stone; it is sometimes easier to make an incision directly over the stone which can then be easily removed.

We then introduce the probe through the incision into the papilla and split the papilla in its whole length. Another way is to introduce the probe into the opening of the papilla and split the intraduodenal part upward.

It is advantageous to unite the mucous membrane of the duodenum with the wall of the common duct immediately after the opening, thus performing an **internal choledochoduodenostomy**. This suture is important also for the control of hemorrhage which may interfere with the further procedure. Thorough control is important to prevent postoperative cholemic hemorrhage which not infrequently occurs in this region. Serious postoperative hematemesis in cholemic patients has been observed because of this bleeding into the duodenum.

The threads of the suture are left long in order to give a good hold for a later procedure. The fingers of the left hand grasp the hepatic duct and the ductus choledochus and work down the stones, while the right hand removes the stones with a spoon near the new opening.

After this manipulation a forceps or small spoon is introduced high up into the branches of the hepatic duct and stones and gravel removed. A small rubber tube or catheter is then introduced into the common duct and warm saline injected by means of a syringe in order to wash out all gravel. It is surprising sometimes to notice how this procedure can expel an unexpected amount of overlooked stones and gravel.

In case an enlargement or induration of the head of the pancreas is found, or if clinically there is a high level of diastasis in the blood and signs of pancreatitis we may also explore the pancreatic duct. However we must admit that pancreatic stones are found rather seldom in this region.*

The duodenal wall is now closed in two layers in the usual way. When no supraduodenal incision has been made and only transduodenal choledochotomy, the abdominal wall can be closed without any drainage.

But when this operation has been performed only because the stones in the papilla could not be removed by the supraduodenal method, it is certainly wise to leave a drain in the abdominal cavity. The wall of the common duct can be closed in two layers as described previously.

Indications and Criticism. This method first described by Kocher offers an excellent exposure of the papilla and we can succeed with a high degree of certainty to remove all stones. This operation has been advo-

* Cases are reported by Moynihan and Mayo-Robson in which they performed an anastomosis between the pancreatic duct and the duodenum, a *Wirsungoduodenostomy*.

cated as a routine by many surgeons (Lorenz) but even the best statistics give a mortality rate from 19 to 30 per cent. In delayed cases the mortality rate surpasses even 50 per cent. The cause for this mortality rate may be the following:

(1) This operation being primarily an additional procedure, when an attempt to empty the ampulla of Vater through the supraduodenal incision has failed, it is obvious that it is rather an extensive operation involving great risk in cases with cholemia and septicemia. General postoperative complications such as failure of circulation, pneumonia, paralytic ileus, hepatic insufficiency and also peritonitis are frequently observed.

(2) Postoperative cholemic hemorrhage into the duodenum and serious hematemesis have been observed as well as cholemic bleeding into the retroduodenal part.

(3) In some cases after the transduodenal choledochotomy incessant vomiting has been observed for a few days following the operation. This may be due to a real mechanical stenosis but it is more likely that injury to the duodenum in this part was sufficient to check peristalsis and normal emptying.

Walzel mentioned a case in which he had to operate for high intestinal obstruction eight days after having performed the transduodenal choledochotomy. He performed successfully an additional gastroenterostomy.

(4) There exists the danger of injury to the pancreatic duct when splitting the papilla. When introducing a probe into the common duct, it is not infrequent that the probe passes into the Wirsungian duct which, in infected cases, may lead to acute pancreatitis. But with or without such a false way of introducing the probe, acute pancreatitis has been observed to follow this transduodenal operation and is often the cause of a fatal outcome.

(5) When providing for drainage through the supraduodenal incision or even after having sutured this incision, the leakage of very active pancreatic juice through the wound which seriously damages healing has been observed.

(6) In badly infected cases in which drainage is unavoidable, leakage through the duodenal suture causes a duodenal fistula with a high mortality rate. Undoubtedly the results are better when this transduodenal choledochotomy for stones in the common duct has been performed as a primary operation without opening the common duct at all. The operation is not so extensive and the mortality rate is lower.

I have always been very reluctant to perform this operation and reserved it only for exceptional cases. Since the development of the ether method, I have given it up altogether.

CHOLEDOCHODUODENOSTOMY (Fig. 154). I should like to describe this operation and discuss its value and indications fully, because it has been

advocated also as an urgent operation for impacted stones in the papilla in which infection was present. It is a so-called short-circuit operation

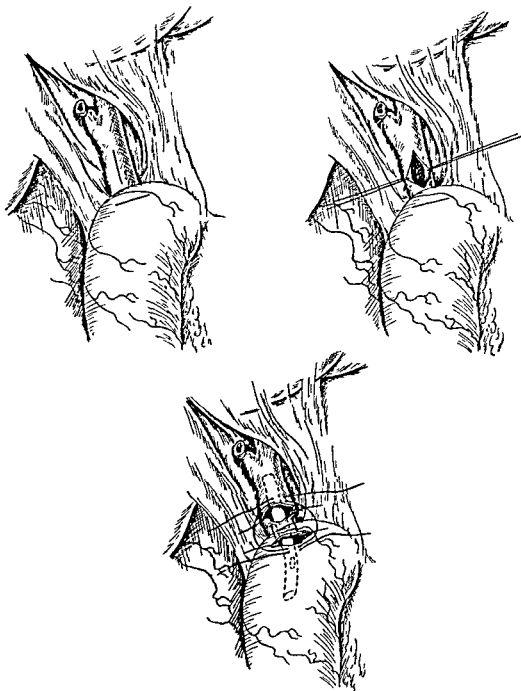


FIG. 154. Choledochoduodenostomy.

and its aim is to avoid the site of the obstruction and to secure bile outflow by another opening between the common duct in its supraduodenal part and the duodenum.

The operation was first performed by Riedel in 1888, but later was popularized especially by Sasse. It is not a very common operation in America or England, is not favored in France, but is especially advocated by Austrian and German surgeons (Finsterer, Floercken). The mortality rate, according to statistical figures, is lower than that of the transduodenal or retroduodenal operation (10 and 20 per cent). The wide opening should allow the passing of stones and gravel from the bile ducts also in the post-operative course. It should secure the outflow of bile by internal drainage, thus avoiding the drawbacks of external drainage. It should avoid also the more dangerous operation on the papilla, when impacted stones in this place could not be removed by the supra-duodenal incision.

It is a short-circuit operation also in cases of mechanical obstruction caused by stricture and compression through the inflamed head of the pancreas or through glands and new growth. Furthermore, it has been performed as a secondary operation for recurrences.

In all operations on the common duct a definite plan of procedure should be made in advance, whenever possible. Hence, when we contemplate a choledochoduodenostomy the common duct should be opened with an adequate incision, low down, in view of the anastomosis with the duodenum which has to be made. Additional care should be taken, when manipulating the common duct and working out the stones, that the wall of the common duct is not damaged. It is sometimes useful to mobilize the duodenum so that the duct can be left in an unchanged position when performing the anastomosis.

But it is not necessary to mobilize the duodenum in all cases and to perform the anastomosis in the retroduodenal part as has been advocated by some surgeons. I am reluctant to perform any mobilization in jaundiced and badly infected cases.

The duodenum is incised in the most suitable way, that is to say, at a place which can be easily approached from the common duct incision. When a longitudinal incision has been made in the common duct, it is best to make a transverse incision into the duodenum. The duodenum is then opened so that the anastomosis is at least one-half or three-quarters of an inch wide. It is very helpful to perform the anastomosis over a rubber drain. The upper end of the tube is introduced into the common duct incision, the lower end into the duodenum. For the anastomosis I prefer fine silk sutures which make only small holes, do not tear so easily and are certainly more reliable. I have never seen any drawbacks. The suture line can be reinforced by the lower part of the hepatoduodenal ligament or, when this is difficult, by omentum. After the suture has been made it is quite easy to push down the rubber drain into the duodenum. Otherwise there is no harm in leaving the drain in the site of anastomosis. It is expelled after a few days with the feces.

In infected cases we may come face to face with a serious dilemma: Leaving a drain means endangering the anastomosis; closure without drainage also involves a certain amount of risk. Leakage through the anastomosis leads to the very unpleasant complication of a duodenal fistula. If circumstances permit we had better close the abdominal wall without any drainage.

Indications and Comments. Advantages: (1) The wide anastomosis not only allows free drainage of bile but also allows stones and gravel to pass freely from the common duct into the duodenum. The internal drainage of bile is advantageous in the sense that bile, which is useful for the digestion of food, especially fatty food, drains into the intestines immediately after the operation.

(2) Statistical figures have proved that the mortality rate is lower than when the retroduodenal or transduodenal operation is performed. This mortality rate is given as 10 to 20 per cent.

Disadvantages: (1) Cases have been reported in which stones, apparently coming from the papilla, have increased their size because of incrustation and have caused an obstruction (Petermann). But this should be a rare occurrence if the anastomosis has been performed widely enough.

(2) In cases of cholangitis, internal drainage through the anastomosis has not the same effect in controlling infection in the bile ducts as has external drainage. I have learned in a remarkable case that the infecting germs draining with the bile into the duodenum may be reabsorbed by the lymphatic vessels or the portal vein, so that septic hepatitis may be carried on in a vicious circle. The following case illustrates this fact:

Mrs. F. G., forty-six years of age, was operated upon on June 25, 1928, for gallstones. She was jaundiced and was in a septic state. Through a costal incision the inflamed gallbladder, filled with stones, was electrocoagulated. The common duct was opened but no stones were found there; the papilla was free. Suture of the common duct and closure of the abdominal wall without drainage were performed.

After an uneventful recovery the patient was perfectly all right until December 20, when she was admitted to the clinic with a severe colicky attack with jaundice. Through the duodenal tube, in which bile was present, *Bacillus coli* and *Streptococcus viridans* could be cultivated. The diagnosis was recurrent hepatitis.

Treatment consisted in washing the duodenum and intravenous injection of glucose and insulin. The patient was sent home in good health. Aside from a slight attack on February 13, she felt well until the middle of April. After a meal of bread and lard a severe colic set in followed by jaundice and alternating temperature up to 103° C. In spite of all medical treatment her condition deteriorated rapidly and she was in a serious septic state.

Another operation was decided upon on May 10. The liver was enlarged and indurated; there was no abscess and no suppuration. The common duct was as wide as a finger. By needling the hepatic duct with a syringe, clear yellow bile was aspirated which was kept for bacteriological examination. No stones were found and the papilla was perfectly patent. There was no stasis whatever in the common duct. Even a thick bougie could easily pass through the papilla into the duodenum. The common duct was sutured again and also the abdominal wall without drainage. From the bile a pure culture of *Bacillus coli* could be cultivated. An uneventful recovery followed.

Treatment consisted in frequent washing of the duodenum by means of the duodenal tube with a 33 per cent solution of magnesium sulphate and the injection of coli serum. Drainage of bile into the intestines was very abundant and the stool was very dark from drained bile. In spite of this energetic treatment no improvement was obtained and a rapid deterioration took place leading to a serious septic state with chills and alternating temperature.

Therefore, a third operation was performed on May 29. The common duct, which was free from all stones, was again opened; and the papilla into the duodenum was also widely opened. A drain was introduced into the hepatic duct toward the liver in order to secure a total external drainage of bile.

The postoperative course was very satisfactory; the temperature became normal and on July 9 the patient was released in a perfect state of health, aside from the liver which was still somewhat enlarged and hard. In the follow-up the patient was seen every year for eight years without having any further attack and the liver became quite normal.

Summary. The patient with a serious hepatitis apparently due to *Bacillus coli* suffered from recurrent acute attacks of septic hepatitis with jaundice. In spite of the fact that no obstruction of the common duct could be found and that drainage into the duodenum was not hindered, the septic state could not be corrected. Only the temporary total external drainage of bile succeeded at once in interrupting the septic state and eventually in healing the hepatitis.

We must assume that the *b. coli* draining into the duodenum had been reabsorbed either by the lymphatic vessels or by the portal vein, reaching the liver again and sustaining in this way a septic hepatitis. Only after this circle had been interrupted by external drainage could the infection be brought to a standstill.

(3) To perform only a short-circuit operation and leave the stones in the papilla entails running a risk, because the stones may also cause an obstruction in the pancreatic duct and interfere with the drainage of pancreatic juice.

(4) Ascending cholangitis has been reported in several cases. This is one of the main objections against the operation.

When patients with a choledochoduodenostomy are given barium meal and lie on their right side, it can be observed that not only the common duct is filled with the opaque liquid but also that it ascends into the small intrahepatic ducts. Therefore, the patient should be advised not to lie down after a meal but rather to sit or to walk, or when lying down to recline on his left side only. Cases of ascending cholangitis are reported by many surgeons. The danger seems to be less when the anastomosis has been performed as wide as possible. There are cases reported (Finsterer) in which after the barium meal all the intrahepatic ducts can easily be filled and in spite of this fact, the patients have been in perfect health for several years. I made the same observation in my own cases.

(5) It must be admitted that shrinking of the anastomosis is observed even when it has been performed one-half or three-fourths of an inch wide, especially in infected cases.

To summarize I would express my opinion that choledochoduodenostomy is a good short-circuit operation but the indications for it are not too many. These indications are all cicatricial stenoses, but I must say that a real cicatricial stenosis is not so frequent as to interfere with the outflow of bile when all stones have been removed. It is a good operation when, in the case of pancreatitis, the swollen head of the pancreas or swollen glands interfere with the free outflow of bile. It must also be considered as suitable drainage treatment in pancreatitis.

The operation has good results at times in the case of dyskinesias after cholecystectomy. But I do not think that this operation should be considered for urgent surgery except in acute cases of obstruction in the papilla as has been mentioned.

In badly infected cases I strongly believe that external drainage is preferable to internal drainage when performing this side-tracking operation. An objection which I have frequently made is that the sudden fall of the back pressure—the maintenance of which I consider of the greatest value after decompression—is a serious drawback to the anastomosis.

In using the technic of external drainage with a watertight tube the back pressure can be maintained and regulated better than in the case of a wide internal fistula without a regulating sphincter.

The objection that the loss of bile through the external fistula causes serious drawbacks is not justified, since the x-ray control by cholangiogram has shown that when using regulated external drainage and the ether method, a sufficient amount of bile passes into the duodenum even after a few days.

The main objection, of course, against choledochoduodenostomy in

acute cases is that the mortality rate is twice or three times as high as when using the ether method.

Ether Method. The ether method is based on the following fact: All gallstones consist of a certain amount of cholesterol which forms their covering. The cholesterol content of the gallstones varies from a few per cent to practically 100 per cent. It can be easily dissolved by ether and we can satisfy ourselves by a simple experiment in a test tube how a gallstone, when treated with ether, starts to crumble after a short time and is transformed into a soft brown mass which can easily pass even a small papilla. Therefore, we may call this process a breakdown rather than a dissolution.

Technic. The patient is operated upon in the manner already described. A costal incision is made; bleeding points are carefully controlled, and the liver and common duct are inspected in order to plan for the procedure. The bladder is emptied by aspiration. In suitable cases subserous cholecystectomy or electrocoagulation is performed in order to preserve the serous layers. This is possible in the great majority of cases. Careful covering of the liver bed should be carried out so that this operative field is perfectly dry and definitely controlled; peritoneal pads should be changed.

After a new walling off of the common duct the hepatoduodenal ligament is divided and bile from the common duct is aspirated with a fine needle on a syringe. A supraduodenal incision is made and all stones which can should be removed. When the papilla has proved to be perfectly free, a gentle dilatation is performed not larger than the size of a pencil in order to avoid cracks which may seriously bleed in cholemic cases. Too wide a dilatation, as advocated by some surgeons, should also be avoided because an undesired reflux of active pancreatic juice into the common duct has been observed.

When the papilla appears to be obstructed and we have to deal with a nonacute case with only recently developed jaundice, that is to say, with a case in a favorable condition, we may extend the probing of the papilla by dividing the lateral peritoneum on the border of the duodenum, gently introducing the forefinger and middlefinger of the left hand behind the duodenum and try to pass the probe again under the guidance of these two fingers. If this trial fails the peritoneum is sewn together and any further forcing of the papilla must be strictly avoided. But whether the papilla has been found free or obstructed, the procedure is the same in all cases and external drainage of the common duct is carried out in order to perform the x-ray control by means of a cholangiogram after a few days. Only this control assures one that no stones are left and the common duct is free.

X-ray control as a routine method in such cases has proved advantageous and is the only way to avoid recurrences caused by stones that

have been overlooked. Indeed this occurrence is much more frequent than we would expect. Recent statistical figures have proved that in 16 to 20 per cent (Mayo, Jung, Brüning) stones are overlooked and that 50 per cent of all recurrences are caused by stones that have been missed (Kirschner's Clinic).

(1) The drain is introduced through the opening in the common duct right down to the papilla, the tip touching the stone. Care should be taken so that the drain does not kink or pass into the duodenum. The opening in the common duct is sewn so as to be watertight or "bile-safe" around the drain which is held in its place by binding the catgut sutures around it. The drain should not be too wide, so that there may be ample room in the common duct for the bile from the hepatic duct to flow alongside the drain.

(2) The hepatoduodenal ligament is also sewn around the drain to carry out an exact peritonization of the operative field. Watertightness is useful for a clear cholangiogram. Other drainage is seldom necessary but there is no harm in introducing a safety drain into the abdomen for forty-eight hours.

(3) Between the fifth and seventh day, according to the condition of the patient, the first cholangiography is performed. The technic is very simple:

The patient is laid on the x-ray table over the Potter-Bucky frame. The bandage is removed and all traces of adhesive on the skin are washed away with benzine. Only the drain is secured with a small piece of adhesive to the skin or better still held in position by an assistant. Twenty cc. of lipiodol, warmed to body temperature, is then slowly injected through the rubber tube into the common duct and the patient is asked to report when he feels undue pressure. In most cases 20 cc. can easily be injected.

Then the first x-ray picture is taken. It shows in many cases that even when the papilla was absolutely impermeable at operation, a small amount trickles into the duodenum. After a few minutes a second x-ray picture is taken. When the result of this cholangiogram is clear, drainage is allowed until the lipiodol has flown out and bile starts to drain again. When stones have been found the ether treatment can be started on the same day.

(4) One must remember that ether boils at body temperature; hence the evaporating ether causes some pressure in the common duct which the patient may feel in the epigastrium. The amount of ether to be used depends entirely on the width of the common duct; in an enlarged duct, 0.5 cc. or even 1.0 cc. can easily be injected, while a small duct may take only a few drops. The bile is first aspirated out of the drain with a syringe in order to empty the duct as much as possible. The syringe is then filled with ether, which is carefully injected drop by drop. The pa-

tient is told to announce when he feels pressure, and when he gives the signal the contents are aspirated again and the pressure immediately ceases.

The injection can be repeated several times in the same way so that a real lavage is performed. Finally 1.2 cc. of liquid paraffin is injected and the drain closed with a clamp. The patient is told to leave the clamp on as long as possible and to remove it only when he feels pressure. When removing the clamp we have immediate evidence whether or not a stasis in the common duct still persists. In the case of stasis, even of a minor degree, the color of the bile is green because of the presence of biliverdin, while in the case of free drainage into the duodenum the bile has a golden brown color.

In some cases adrenalin and atropine, or 0.5 per cent solution of percaine can be added to facilitate relaxation of the sphincter of Oddi. This procedure may be repeated two or three times a day according to the particular case. An injection of 50 cc. of a 33 per cent solution of magnesium sulphate can also be given through a duodenal tube once or twice a week alternately with olive oil.

The time that the patient can stand the occlusion of the drain sometimes increases very rapidly, the papilla becoming more and more permeable. After a week's treatment a new cholangiogram is taken. The patient can leave the hospital after two or three weeks and may be treated as an out patient or, if necessary, at home. The drain is removed when the cholangiogram shows that the whole common duct and the papilla are empty. The small fistula will always close within forty-eight hours.

The surgeon should always bear in mind that the ether method is a conservative form of treatment, and that the time required for dissolving stones and emptying the papilla varies considerably. In some cases we need a few days and in others as long as eight weeks.

For example, at the operation upon a man, aged sixty-four, an absolutely impermeable papilla was found which could not be emptied even after six weeks of ether treatment. I was already considering a new operation with a transduodenal opening. Owing to the tendency of the obstruction and the unusual failure of the ether treatment, I even suspected a carcinoma of the papilla. Fortunately, I had to postpone the operation for another week and meanwhile continued the ether treatment. The next cholangiogram taken a week later showed that the ampulla was empty. I followed up this patient for five years. He remained in perfectly good health and had no further trouble.

It is important, therefore, not to become discouraged, even if after treating a patient for several weeks the cholangiogram shows that the ampulla has not yet been entirely emptied. The time required for dissolv-

ing the stones or transforming them into a pulp that will pass through the papilla depends on many factors.

I have sometimes seen stones hidden in inflamed and swollen crypts; such a situation will delay their dissolution; but the fact that I have finally met with success in all thirty-eight cases, including some obstinate biliary fistulae, and that since 1932 I have not had to perform any operations such as retroduodenal or transduodenal choledochotomy, makes me confident that we can rely on the method, and I think we should consider a second operation only after long and thorough ether treatment has failed.

Indications. The ether method deserves in my opinion wide use in urgent surgery for obstructed stones in the ampulla of Vater. There is no doubt that this kind of procedure involves the lowest operative risk. When performing an electrocoagulation of the gallbladder and sewing a small rubber drain into the supraduodenal choledochotomy incision, through which accessible stones have been removed, the whole operation can be performed in thirty minutes.

This simple procedure has reduced the mortality rate to 5 per cent in these serious cases, instead of 20 to 50 per cent. Two patients have died out of forty with signs of progressing hepatic insufficiency before the treatment could be started. Both patients—one of whom was a doctor sixty years of age—suffered for weeks from obstructive jaundice with cholangitis, could not recover from their drowsiness after operation and died in hepatic coma.

The results so far have been very encouraging. Out of thirty-eight cases in which the ether method has been used the papilla became permeable in *all* cases. Obviously we must always keep in mind that we may have to consider a second operation when for some special reason the ether treatment should fail. But even when we have to do so the advantages of this two-stage operation for obstructed stones in the papilla are evident. At the second operation we operate upon a patient in quite a different state: there is no jaundice any more, septicemia is overcome and hepatic function is considerably improved.

On the other hand, the cholangiogram presents definite evidence about the conditions in the common duct: stones, cicatricial stenosis, pancreatitis, etc. When stenosis in the papilla is the cause of incomplete emptying, we may consider a choledochoduodenostomy as the second operation. But as a matter of fact these considerations so far have been theoretical only. I was not forced to perform a second operation at all in any one of my thirty-eight cases. The follow-up has shown that only one had recurrence of gallstones.

The patient, a man aged forty-three, was admitted to the hospital in December 1935. He had had jaundice in 1918 for the first time and

afterward had a sensation of pressure frequently in the upper portion of the abdomen. Six months before being admitted to the hospital he had developed pain in the upper part of the abdomen. Cholecystography showed no filling of the gallbladder but no special treatment was given. Later colic set in again and was followed by jaundice. On admission to the clinic he was very ill with severe jaundice and multiple cholemic subcutaneous hemorrhages; any pressure on the skin produced bruising. His circulation was poor and he had high alternating temperature with frequent chills. The urobilinogen test was green instead of red which I have always found to be a sign of a badly damaged liver.

Operation was performed on December 9, 1935. The liver was enlarged and friable and the gallbladder badly inflamed containing purulent mucus and small stones. After electrocoagulation (mucoclasia), the common duct was incised and abundant mucus and stones removed. The stones reached high up into the hepatic ducts. They were removed as far as possible and the operation was ended quickly because the condition of the patient had become very poor. A rubber tube was introduced into the common duct and sewn water tight and the abdominal wound closed. After the operation glucose and calcium were injected by intravenous drip. Parathormone was administered and gelatin injected. Prontosil and cardiac stimulants were also given.

The patient improved gradually and on the twelfth day after the operation the ether treatment was started. He had chills again with a rise in temperature several times a day. The wound had to be handled very carefully because any movements of the drain caused immediate bleeding. The appearance of the bile gradually improved and eventually became brown and clear. There was never any serious hemorrhage. The patient had fever and chills until January 11, 1936.

The progress of treatment was controlled by repeated cholangiography. On March 6, 1936, cholangiography proved that the papilla and the common duct were entirely free. On March 8, he was discharged from the clinic in good condition but the liver remained enlarged apparently due to cirrhosis.

Between 1936 and 1938 his health remained good but in 1938 he was again ill and as far as I can ascertain was operated upon somewhere else for a "recurrence of gallstones."

It is not quite clear whether some gallstones remained in the intrahepatic duct and later descended causing a new occlusion of the papilla, or whether only his biliary cirrhosis made further progress. As the cholangiogram taken before removing the tube showed no stones, the common duct and the papilla were certainly free and the fistula closed in forty-eight hours and remained closed for two years. Therefore, it might be that the recurrence was due only to a recurrent hepatitis with jaundice.

PRE- AND POSTOPERATIVE TREATMENT. As mentioned before there is, as a rule, not much time left for preoperative treatment in urgent surgery. But even during twelve to twenty-four hours which may be at our disposal a good deal for the improvement of the general state and especially the function of the liver can be done.

(1) *Liver and Gallbladder.* The acute pathological trauma which takes place in the hepatobiliary system causes an interference with the function of the liver almost always. Leaving aside injuries caused by external force, the acute inflammatory process is usually in the foreground.

It is important to bear in mind that in every acute inflammatory process in the abdominal cavity a certain amount of immunity and self-defense gradually develops. The effect of this self-defense is the **localization** of the process. Within this area of localization immunity is of great value for the healing process. Therefore, all means which would favor this process of localization should be applied. A **slowing down in the progress of the inflammation** is the best means of developing self-defense and immunity. Slowing down of such an acute inflammatory process can be compared in its effect to active immunization by the injection of less virulent germs.

The best means at our disposal for slowing down an inflammatory process is the **application of ice**. Therefore, I think it is advisable that during the whole time which we can spare in postponing the operation an **icebag** should be applied at the region of the liver and gallbladder. Application of warmth accelerates the development of inflammation and suppuration. The process of self-defense and immunization always takes a certain time. Therefore, I believe hot applications should be **strictly avoided** in acute cases.

(2) *Edema of the Liver.* The most frequent response of the liver in hepatobiliary diseases, especially in acute infection, is a marked edema comparable to edema of all infected tissues. This condition of the liver causes the organ to be considerably enlarged, soft and of a frail consistency. It reaches sometimes more than a hand's breadth down under the costal arch. This edema is characterized by its fleeting character. It is the first stage of hepatitis and is always accompanied by a certain amount of functional weakness. The combating of this acute edema is a very important task in the pre- and postoperative treatment of the liver, and it can be achieved quite easily by osmotic therapy, namely, dehydrating the liver by intravenous injections of hypertonic solutions.

We start this dehydrating therapy immediately by injecting 20 cc. of 50 per cent glucose combined with calcium three times a day. The effect of this procedure is often striking. The enlargement of the liver diminishes and undue pressure may relax. Another favorable effect of the glucose injection is the replenishing of the glycogen storage.

(3) *Hemorrhagic Diathesis.* Postoperative hemorrhage is most serious

and is frequently the cause of a fatal outcome, occurring even days after an otherwise successful operation.

A great deal of research has been done concerning the causes and conditions responsible for cholemic patients bleeding after operation. We have some tests which to a certain degree enable us to find out the diathesis of these patients before operation. This is certainly important for our pre- and postoperative treatment, but I cannot agree that when these tests reveal a great possibility of dangerous postoperative hemorrhage that this should be considered a contraindication to operate, as believed by some surgeons. We have to consider the fact that these patients *require* an urgent operation, that their condition deteriorates from day to day and that a fatal outcome will not be postponed by any other treatment than that of operation.

Therefore, I believe we should give the patient a chance and run the risk of an operation despite what these tests may have shown. In my opinion the practical value of these tests lies not in their interference with our decision as to whether or not we have to operate, but rather in the improvement and modifying of our technic, in the indications they suggest for the simplest way to perform the operation, in the choice of a proper anesthesia and in the thoroughness of our pre- and postoperative treatment.

Two factors must be considered important in order to explain this hemorrhagic diathesis: (1) An increased permeability of the capillaries not only for fluid but also for corpuscular elements, such as blood cells; (2) disturbance in the physiology of blood coagulation. A simple test for these conditions can be made in the following way (Ivy):

The blood pressure cuff is applied to the arm, the pressure raised to 40 millimeters of mercury and maintained at that point for one minute. Then a puncture of 2.5 mm. is made on the forearm with a mechanical stilette. At a constant pressure of 40 mm. the upper limit of bleeding time in the normal individual is approximately 240 seconds. In jaundiced patients inclining toward hemorrhage, the bleeding time can be prolonged considerably.

In the same way the permeability test can be carried out. When the tourniquet is applied for a certain length of time, multiple petechiae appear on the skin and, when knocking with a percussion hammer, subcutaneous hemorrhages also appear. This test, which is significant for the vulnerability of the capillaries and larger vessels, is obviously a real test for the reaction of tissue against injuries.

A test for the clotting process is achieved by investigating the prothrombin, antithrombin and fibrinogen content of the blood. When multiplying prothrombin by fibrinogen and dividing it by antithrombin, an index is obtained which is normally 0.7 or above. A lower index indicates a certain hemorrhagic tendency.

The sedimentation rate also can be a useful test. A very rapid sedimentation of the red blood cells indicates hemorrhagic diathesis.

PRECAUTIONARY MEASURES. (1) The principal rule to be considered is that any delay and undue postponement of operation in case of obstructive jaundice should be strictly avoided. The old rule to wait for about six weeks is a baneful mistake.

I have already discussed the way which has proved most satisfactory how to manage the patient during the first days following the attack. In infected cases the operation should be performed as an urgent operation within twenty-four hours; in noninfected cases a decision must be made within a week's time at the most.

(2) *Surgical Precautions.* Improvement of surgical technic has been valuable in the prevention of postoperative cholemic hemorrhage. The greatest thoroughness should be employed in the control of bleeding, in the avoiding of any damage to the liver bed and in the covering of the whole operative field with serous tissue (subserous excision, electro-surgical operation).

Only the simplest operation should be performed in poor risk cases; the ether method is to be used in the postoperative treatment for impacted stones. The danger of hemorrhage is lessened in this way to a high degree.

BIOLOGICAL METHODS TO PREVENT POSTOPERATIVE HEMORRHAGE. Two determining factors must be considered: (1) the pathology of coagulation, and (2) the increased permeability of the capillaries.

Pathology of Coagulation. Research work in recent years has contributed a great deal toward the understanding of this factor. The tendency to bleed in hemorrhagic diathesis depends definitely on a deficiency of plasma prothrombin. This prothrombin must be considered a physiological entity rather than a chemical substance (Pateck, Taylor). By omitting certain fat-soluble substances in the diet, diathesis can be produced experimentally while the plasma prothrombin content drops. This diathesis can be stopped and relieved, on the other hand, by giving certain sterols, commonly known as vitamin K.

The prothrombin index can be increased by vitamin K which is found in prothrombin itself. Vitamin K is a fat-soluble substance which is only reabsorbed from the intestines with the aid of bile and bile acids.

The whole process seems to be reasonably clear and so also the rules for therapeutic measures. In case of obstructive jaundice no bile acids reach the intestines, reabsorption of vitamin K from food is hindered, prothrombin in the plasma drops and the clotting mechanism of the blood is seriously damaged.

As for the treatment vitamin K in capsules with desoxycholic acid or bile is given. Results are encouraging.

But with due regard for the great practical value of this research work,

we must nevertheless admit that hemorrhagic diathesis and postoperative cholemic hemorrhage are not fully explained by the clotting factor alone.

Increased Permeability of the Capillaries. In my opinion the second factor, i.e., the increased permeability of the damaged capillaries has been neglected too much apparently because of the discoveries of vitamin K and its influence on prothrombin and the clotting mechanism.

Muscular hematomas, the bleeding from noninjured surfaces as well as the huge fatal bleeding into the intestines, which we observe during the postoperative course, are due more to the increased permeability of the damaged capillaries. And this is what we have to fear when operating. This permeability is the cause of the petechiae which can be produced in the skin of the whole arm as a test when applying the blood pressure apparatus.

In tightening the wall of the capillaries lies the value of calcium therapy which has been challenged and disputed in the last years as having no effect whatever on cholemic hemorrhage. This opinion was based mainly on experimental findings which showed that here was no marked increase of the calcium level in the blood nor any influence on bleeding or coagulation time or on the prothrombin index.

But, as already mentioned, in my opinion the value of calcium therapy, which in clinical work has impressed me several times especially when combined with parathormone, lies not in its influence upon the coagulation process, but in the fact that the intravenous calcium application combats the pathological permeability of the capillaries.

I use calcium therapy in pre- and postoperative treatment as a routine. Twice a day the patient receives intravenously 10 to 20 cc. of a 10 per cent solution of calcium gluconate. In addition intramuscular injections of parathormone (Lilly) 0.5 cc. are given daily. I do not think it wise to condemn the application of calcium as useless in regard to the capillary factor in cholemic hemorrhages.

SUMMARY. The rules for combating cholemic hemorrhages may be established as follows:

(1) *Surgical Precautions.* Simplification of procedure in cases of hemorrhagic diathesis.

(a) Avoidance of retro- and transduodenal method for impacted stones in the ampulla by applying the ether method.

(b) Careful surgical control of hemorrhage when operating; strict avoidance of tamponade.

(2) *Biological Method.*

(a) Application of vitamin K to increase the prothrombin index.

(b) Application of calcium and parathormone to combat increased permeability of capillaries.

(c) Hemostyptic serum (Parke, Davis).

(d) Blood transfusion.

In serious cases a blood transfusion should be applied before and after the operation. But it must be said that in spite of all enthusiasm the special results in preventing cholemic hemorrhage are not absolutely convincing, and in cases of serious hepatic insufficiency there is a certain factor of doubt as to whether a blood transfusion means additional work for the liver or not.

ACUTE INSUFFICIENCY OF THE LIVER FOLLOWING DECOMPRESSION. The precautionary measures have been discussed fully when dealing with the question of drainage. Maintenance of the back pressure in the technic of drainage has proved an important factor.

RESPIRATORY TRACT. When there is an acute or subacute infection in the respiratory tract, the operation should be postponed if possible. But for obvious reasons in urgent surgery we can postpone the operation only within certain limits. We are often forced to make the best of the patient's condition. Painting of the throat with iodine and camphor solution is always advisable and should be carried out at once. For patients with bronchitis an injection of the soluble quinine derivatives has proved to be by far the best (solvochin and camphor in ether). Hot applications round the chest, inhalants and expectorants of all kinds are advisable together with breathing exercises immediately after the operation with the help of carbon dioxide.

Spinal anesthesia is in all cases of hepatic insufficiency the anesthesia of choice; it is also the best when the patients are endangered by the conditions existing in their respiratory tracts. But it has been proved that an additional general anesthesia with gas-oxygen has some advantages over the pure spinal anesthesia, because artificial rhythmical ventilation of the lungs during operation has its definite value. I never use local anesthesia in jaundiced patients; hematomas at the place of the injection have been observed. When there is an infection of the lungs, spinal anesthesia with gas-oxygen is also preferable.

During the operation any pulling on the liver and the diaphragm should be strictly avoided. The incision must have ample exposure so that we can operate *in situ* as gently as possible without changing the position of the liver. It is a well known and important fact that in all operations upon the liver some damage is easily done to the diaphragm and that the lower lobe of the lung is in real danger of becoming congested. It may be followed by massive collapse and consequent pneumonia. The same holds true on the left side when for instance a splenectomy is performed.

The complications in the right lower lobe following operation on the hepatobiliary tract may form a real danger in the postoperative course and increase the mortality. On the other hand, if we bear this danger in mind we are in a position to overcome it to a considerable degree with our surgical precautions.

Belonging to these surgical measures in lung complications I also include the careful control of hemorrhage. When we rely upon clotting only for the stoppage of hemorrhage in smaller vessels without closing the vessels by ligatures, these clots are more likely to become infected. We must realize that embolism of such infected blood clots may cause small spots of infection in the lungs and these infected spots may give rise to bronchopneumonia and a spreading of infection.

This modus of lung complications is certainly most important and is an explanation for the statistical figures, which prove that when applying local anesthesia and using the same technic lung complications are not lower than when using general anesthesia. A correct surgical technic and especially the careful control of hemorrhage must be considered as the most important precautionary measures against lung complications.

HICCUGH. Hiccough can be one of the most tiresome and tenacious postoperative troubles. The best measure to counteract this condition so far is the inhalation of carbon dioxide. It was successful in cases in which all other methods had failed; therefore, I cannot advise anything better. In some cases this embarrassing reflex could be stopped by painting the mucous membrane of the nose with novocain-adrenalin.

CARDIOVASCULAR PREPARATION. Some physicians advocate treatment with digitalis or other cardiac stimulants before operation at all times. We are rather reluctant to do so and have always found, in collaboration with our consulting physicians, that routine preoperative cardiovascular treatment is not necessary or even advisable. We limit cardiac treatment to the demands of the special case and must say that, according to our experience, the strain of the operation on the patient's heart has been rather overrated.

If we analyze all cases with a fatal outcome, we can scarcely find one that is directly due to overstrain of the heart and circulation immediately after operation. Cases of acute cardiovascular collapse, however, occur following removal of an obstruction of the common duct followed by sudden decompression, the latter being the usual cause.

The most striking case that I have observed was that of a woman, seventy-three years of age, with cholemia, septicemia and diabetes due to chronic pancreatitis. Before the anesthetic could be administered she suffered a serious circulatory collapse on the operating table and for one hour we had to apply all kinds of stimulants before we could start. The operation which was quickly performed under spinal anesthesia (mucoclasis of the empyematous bladder and removal of a common duct stone) was performed without the slightest change in the patient's circulatory condition. Aside from the heart trouble from which the patient has suffered for years, she now enjoys a satisfactory general state of health eight years after the operation and at the age of eighty-one.

The cardiovascular disturbance in the postoperative course, starting usually on the third or fourth day, is in most cases purely secondary, due to absorption of toxic products from the operative field. These toxins are frequently proteinogenous products of histamine character, but they may also be bacteriogenic toxins, all of which damage the peripheral circulation and cause lowering of blood pressure.

This toxic insufficiency of the peripheral circulation is best combated by an intravenous drip infusion which, according to my experience, has proved very satisfactory in the detoxication of the patient and the improvement of the whole peripheral circulation. The choice of the solution is important. I often apply glucose and saline, but have found a mixture of citrate salt solutions in addition very helpful.

The patient should not perspire, even when a drip infusion is continued for four to five days. Perspiration is a sign that the solution has not been retained in the blood vessels and that it permeates the capillaries. If there is any inclination to perspire, a 1 per cent calcium citrate solution should be added to the intravenous infusion. Parathormone injections (5 units), which retain the calcium level in the blood, have also proved useful. According to the individual case, strophanthine (1/60 of a grain) can be added as well as digitalis preparations. But this is not the place to discuss the special indications for digitalis therapy in the postoperative treatment.

POSTOPERATIVE BOWEL MOTION. I am not in favor of forcing the bowels to move during the first days after the operation. For twenty-four to thirty-six hours the bowels are practically always paralyzed but this causes no harm and the rest provides the best conditions for abdominal wounds to heal and for inflammatory processes to localize. When the conditions in the abdominal cavity appear normal, the intestines start to move almost without help. *On the other hand, when there is an uncontrolled inflammation, any movement excited by forcible means (purgatives, irritants, etc.) may cause a spread of the inflammation.*

The only thing I do is to give an injection of 20 cc. of glycerin into the rectum but only when the bowels have already started to move and the rhythm is still disturbed and the purpose of the motion not achieved. I do not give any oral purgatives as a rule before the fifth day. Should peristalsis be delayed in spite of the absence of any peritoneal irritation, 1/60 grain of physostygmmin or prostygmmin intravenously has always a reliable effect.

Abdominal Complications

PERITONITIS. I must emphasize again that peritonitis following a gall-stone operation has become a very rare complication with our modern technic. As one of the main points of the technic I should like to men-

tion (a) limitation of the operation to the field of local immunity; the costal incision has proved to be a great help for this purpose; (b) electrosurgical operation in all infected cases and exact peritonization of the operative field.

But in seriously infected and bad risk cases I use some precautionary measures which I think are of some value. These consist (a) of an intramuscular injection of 20 cc. of coli serum for the first three days following the operation at the latest. Then these injections must be stopped because of anaphylaxis. I had cases in which this coli serum had a convincing effect. So I am always inclined to give this injection in suitable cases. (b) The administration of 4 gms. daily of sulphanilamide derivatives for the first three days.

ACUTE DILATATION OF THE STOMACH. This is a very serious complication which has been observed on several occasions after an operation on the biliary tract. It is nothing else than a high ileus with vomiting of huge masses from the stomach, causing a serious and quick exhaustion of the patient.

In my experience the padding with swabs, which was formerly very common, was to blame in the first place. I have not observed a single case since I have abandoned this method of tamponade, but I have seen some cases during consultation with other surgeons. Removal of the padding has had an immediate effect. Continuous suction of the stomach contents should be administered.

LEAKAGE OF BILE. This complication used to be very common after a classical cholecystectomy. Leakage of bile through the drain was so frequent that it was considered natural. It was assumed that this leakage came from the cystic duct. With the development of the exact peritonization in the electrosurgical operation this complication can be practically excluded so that the closure of the abdominal wall without drainage may be performed in most cases. I believe that this leakage of bile observed so often formerly came rather from the injured liver bed than from the cystic stump.

FISTULAE. A persistent bile fistula is a certain sign of an obstruction in the common duct usually caused by a stone which had been overlooked. When the outflow through the common duct into the duodenum is free, a leakage of bile following the removal of the drainage stops within a few days. When, however, a stone has been overlooked in the common duct, it causes a permanent leakage of bile.

We are now in a position to deal with such a fistula without re-opening the abdomen in the following way:

We introduce a small rubber drain through the fistula. This rubber drain finds its way easily through the formed track into the common duct and down to the point of obstruction. Then an x-ray picture by

means of the injection of lipiodol (cholangiography) is taken. This picture reveals the cause of the obstruction and hence the cause of the bile fistula. Invariably, the presence of stones will be demonstrated. When stones have been found, the ether treatment is performed. I have succeeded in cases of such fistulae lasting for months in freeing the papilla by dissolving the stones sometimes after a week or two.

When by means of cholangiography the papilla has proved to be free, the drain can be removed and the fistula closes within forty-eight hours.

DUODENAL FISTULA. A duodenal fistula is observed (a) in the case of an accidental injury during operation; (b) when perforation of the gallbladder into the duodenum has occurred and when the hole in the duodenum has not been closed carefully enough or has reopened again after removal of the gallbladder. This occurs principally when a drain has been left too near to the duodenal sutures; and (c) when performing an anastomosis between the duodenum and the gallbladder (cholecystoduodenostomy) or an anastomosis between the common duct and the duodenum (choledochoduodenostomy). Such leakage from the duodenum must always be considered a very serious complication leading to cachexia within a short time.

A characteristic clinical sign following leakage from a duodenal suture line is the fall of temperature to 97° F. and below. The skin becomes irritated very soon because of the activity of the duodenal juice. Every effort, therefore, must be made to deal with such a fistula. The measures are:

General. The oral intake of food or liquid should be restricted or avoided for a few days. The patient's need for food must be met either by intravenous drip infusion, a measure which always should be taken in such cases, or by rectal drip infusion.

Tamponade. Tamponade of the wound with gauze soaked in simple olive oil should always be tried.

Suitable Protection of the Skin. Charcoal application as an absorbent, beside the protection of the skin by suitable ointments, has proved helpful. In serious cases with a great loss of duodenal juice and fluid a jejunostomy is sometimes the only measure which may save the patient's life.

POSTOPERATIVE HERNIAE. It is well known that postoperative herniae are especially frequent when operating for gallbladder diseases. The median and the old Kocher and Kehr incisions were most likely to be followed by incisional herniae especially when not only drainage but also padding had been employed. The frequency of such herniae, according to statistical figures, is as high as 15 per cent.

But not only has this regular hernia been observed but also a paralysis and atrophy of the abdominal muscles due to injuries at operation to the

sixth to ninth costal nerves. This may lead to a weakness of the abdominal wall on the right side resulting in a dome-like contour of the abdomen.

This accident is not only a great annoyance to the patient but is also most difficult to repair at a second operation. I must say here that since the development of the costal incision, which I have employed in more than 1,000 cases, I have not observed a single postoperative hernia, even when infection of the wound took place. The same results with this incision have been reported by all surgeons who have adopted this method (Stegemann in 700 cases, Usadel, Orator).

THROMBOSIS. Thrombosis in the veins of the lower extremities is to be feared during the postoperative course. The relative frequency with which such thrombosis occurs has more general than special reasons. In the majority of cases patients with gallstones are adipose women with a history of pregnancy. Varicose veins in the calves are frequent. Preventive measures in poor risk cases, especially with a history of previous thrombosis, should always be carried out. Both legs should be elevated and massaged upward. This should be carried out daily after the operation. I have the impression that these precautionary measures lessen the frequency of thrombosis. But I must say that in some cases I have noticed pain in the veins after such a massage which induced me to stop it. Apparently there was an irritation of previously inflamed veins. Embolism seems to be very rare in gallstone diseases when the modern technic is employed. I have not lost a single patient from embolism in more than 1,000 cases.

TREATMENT OF THROMBOSIS. When a thrombosis in the calf appears, the limb should always be fixed on a splint and elevated. Ichthyol ointment produces the best results.

In conclusion of this chapter we believe that a table of classification and causes of jaundice may not be out of place.

JAUNDICE

(1) Obstructive	{	of intraheptic ducts	{	Cholangitis
				Stones
				Abscesses
				Tumors
	{	of extrahepatic ducts	{	Internal
				Obstruction
				{
				Stones
				Spasm
				Tumor
				Parasites
				{
				Glands
				Abscesses
				Pancreatitis
				Tumors
				(Carcinoma)
				(Sarcoma)

(2) Hepatitis	(a) Auto-intoxication	{ (1) Catarrhal jaundice (simple acute hepatitis) (2) Acute atrophy of liver (3) Cirrhosis
	(b) Infectious Diseases	{ (1) Septicemia (Strepto-, Staphylococci pyemia) (2) Scarletina (3) Pneumonia (4) Typhoid fever (5) Weil's disease (6) Syphilis
	(c) Poisons to Liver Cells	{ (1) Arsenic (2) Phosphorus, etc.
(3) Hematogenous	(a) Congenital Hemolytic	
	(b) Hemolytic Poisons	{ Tolylendiamine Unsaturated fat acids Bothriocephalus
	(c) Pernicious Anemia	

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Chapter XI

Urgent Surgery of the Spleen

By JULIUS L. SPIVACK

Conditions which demand urgent interference are: (A) Injuries of the spleen; (B) torsion of the splenic pedicle; (C) abscess of the spleen; and (D) cysts of the spleen.

INJURIES OF THE SPLEEN

Injuries of the spleen in the great majority of cases are traumatic in origin; however, sometimes, they are of a spontaneous nature.

Traumatic injuries are divided into subcutaneous and open wounds. Subcutaneous injuries are those which occur without external signs of injury and may produce either contusion of the spleen or its rupture. Open injuries are those in which an open wound of the abdominal wall is present in addition to injury of the spleen.

Subcutaneous Injuries

This type of injury may affect the parenchyma only, not affecting the capsule, and it is known then as *contusion* of the spleen; or it may also tear the capsule and produce more or less extensive damage to the parenchyma. This latter condition is known as *rupture* of the spleen.

ETIOLOGY. Subcutaneous injuries occur in any age and in both sexes. In cases in which normal spleens are ruptured they are due mostly to automobile accidents and for this reason occur most frequently in persons who are subject to the hazards of everyday life. These accidents are also the reason for rupture occurring more frequently in the male between the ages of 20 and 40. However, it may occur at any age. The youngest case reported was that of a newborn infant who was dropped on the floor immediately after delivery.

In a pathologically altered spleen, which is observed in patients suffering from typhoid fever, relapsing fever, acute sepsis, splenic anemia and so on, it may be enlarged, the capsule tense and, therefore, the spleen can be ruptured easily even after insignificant external injury, such as a moderate blow on the left side or even on the right side. In countries with moderate climate where the inhabitants have normal spleens, the latter rupture only when violent force is applied, such as occurs in automobile accidents, kicks by a horse, falls from great heights, powerful blows, etc.

INCIDENCE. Injuries of the spleen are not very frequent. According to L. T. Wright and A. Prigot, out of 20,000 cases of traumatic injuries admitted to Harlem Hospital only thirty persons sustained injury to the spleen, which is in a ratio of 1:666.

However, if in case of injury an abdominal viscus is injured, the probability that it will be the spleen is very great. As a matter of fact, it is involved more frequently than any other abdominal viscus; some authors claim that the spleen is injured in about 48 per cent of cases in which an abdominal viscus is injured; next in frequency is the liver, which is injured in about 29 per cent of cases, intestine in 18 per cent, mesentery in about 3 per cent and the pancreas in about 2 per cent. However, other statistics do not give such a high percentage for spleen injuries as compared with injuries of other abdominal organs. Lipkin, in his review of seventy-four cases of trauma of the abdomen, mentions that in fifty-five cases only one viscus was injured and in nineteen cases there were more than one viscus involved. Of the seventy-four cases of injury, the spleen was injured eighteen times, or 24 per cent, of which ten cases were isolated injuries and eight cases of combined injuries. According to his statistics, injuries of the bowel occupied the first place (twenty cases) and were followed in order of frequency by injury to the spleen (eighteen cases), kidney (eighteen cases), liver (nine cases) stomach (eight cases), diaphragm (six cases), urinary bladder (four cases), etc.

AGE. It occurs in any age, but most frequently in children up to the age of ten and, due to occupational hazards, in adults between the ages of twenty and thirty. About 30 per cent of all cases of injury to the spleen occurs in children before the age of ten, and in 20 per cent in adults between thirty and forty years of age.

It may occur as an isolated injury or it may be associated with injury to other structures such as fractured ribs, ruptured liver, contused kidney, broken extremities, ruptured urinary bladder and so on.

PATHOLOGY. Primary injury of the spleen may be grouped into three classes:

1. *Minor Superficial Capsular Rupture or Slight Parenchymal Contusion.* In this type there are small tears in the capsule; the parenchyma is bruised in a slight degree. It may involve the convex or the concave surfaces. In case of injury to the convex surface the bleeding is insignificant but it may be considerable if the concave surface is involved in the proximity of the large blood vessels or its branches. These injuries tend to heal spontaneously.

2. *Subcapsular Hematoma without Rupture of the Capsule.* In this group the capsule is not ruptured. The parenchyma is injured first, producing hemorrhage; blood accumulates under the capsule; with further effusion of blood the hematoma enlarges increasing the tension of the

capsule. The increased mass of blood may remain stationary or break through the capsule, thus causing a severe hemorrhage, or it may destroy the parenchyma of the spleen producing a "blood cyst." The latter has the appearance of a purple lump on the surface of the spleen. In exceptional cases the effused blood destroys all of the parenchyma and the entire spleen is a "blood cyst." Such a case was reported by Felix Lejars.

3. *Capsular and Parenchymal Rupture with Perisplenic Hematoma.* This type is the most frequent and occurs at least in half of all cases of injury to the spleen. It may be of any size, shape and depth, and may be localized in any portion of the spleen. The blood escapes through the capsular opening into the free peritoneal cavity. One of the most frequent ways in which the hemorrhage stops spontaneously is by the omentum reaching the rent in the capsule and plugging it.

Another possibility of preventing the escape of blood into the free peritoneal cavity is the location of the spleen in relation to the surrounding structures. This relation permits the escape of blood from the intracapsular to the extracapsular space, but prevents it from going freely into the free abdominal cavity and keeps it in a circumscribed position in the form of a perisplenic hematoma. This potential space is bounded on all sides by structures easily displaced, so that it can contain a great amount of blood. It is bounded above by the left half of the dome of the diaphragm, below by the splenic flexure of the colon and by the transverse mesocolon, medially by the stomach, laterally and behind by the left hypochondrium, and in front by the omentum and anterior abdominal wall. If the hematoma develops slowly, adhesions are formed between the omentum and stomach and between the omentum and anterior abdominal wall, while the coils of the small intestine and the portions of the transverse colon stick together in the vicinity of the hematoma. In this manner a fairly closed cavity is formed adjoining the spleen, which serves as a seat for the hematoma and prevents flooding of the general peritoneal cavity with blood. However, only in a few cases does the hematoma reach a large size.

The fate of this hematoma is as follows: (a) Complete absorption, (b) encystment. The peripheral part of it becomes fibrous and the center remains serous, sero-sanguineous or sanguineous. (c) The serum is absorbed and the solid portion of the clot persists; it becomes progressively harder and finally is transformed into a mass of hard, greyish-white tissue which is described under the name of "fibrinous tumor of the spleen." (d) Suppuration of the hematoma; this, however, is a rare complication. (e) Rupture of the capsule with ensuing hemorrhage.

SYMPTOMATOLOGY. Subcutaneous injury of the spleen does not give any pathognomonic symptoms. In case of slight injury it may never be detected and only while examining the abdomen for some other condi-

tions or at autopsy may one find a healed scar of the spleen. In case of severe injury of the spleen the patient may show symptoms of hemorrhage and shock and may die before symptoms arise which will point to the real injury.

If we exclude these two extremes, all cases of injury to the spleen will show symptoms either of shock or of hemorrhage in which there is nothing pathognomonic for spleen injury. Some of the symptoms become intensified and others make their appearance only after a few hours and even days which lead one to surmise that injury to the spleen has taken place. The most conspicuous symptoms of a ruptured spleen are:

1. *Shock.* This symptom is present quite frequently but not constantly. It is manifested by its usual signs: pallor, cold sweat, peculiar expression of the face, small and rapid pulse, rapid respiration, etc.

2. *Pain.* This is quite a constant symptom although it is not always present. The pain is usually sharp and felt in the left upper quadrant, but it may be present also in any other region such as the epigastric, right hypochondric, umbilical or right or left iliac regions. In some cases the patients complain of pain in the left shoulder (Kehr's sign). Quénu attributes this sign to the irritation of the diaphragm by blood clots which transmit the irritation through the phrenic nerve to the shoulder region.

3. *Dyspnea* is a frequent symptom. This may be due to diaphragmatic irritation, to injury of the chest wall or to the loss of blood.

4. *Tenderness and rigidity* of the abdominal muscles are usually early symptoms. Rigidity at first is localized, usually in the left upper quadrant; later it becomes generalized and extremely pronounced and may actually reach a board-like degree. However, rigidity may be absent even in cases of a total rupture of the spleen; usually it is present in about half of all cases of a ruptured spleen.

5. *Blood pressure and pulse pressure* maybe normal or lowered. Almost 80 per cent of patients with associated lesions show a lowered blood pressure and an accelerated pulse rate, whereas patients without associated lesions present in the majority of cases a normal blood pressure and normal pulse rate. This may serve as a valuable diagnostic point as to the extent of injury; in other words, it may help to decide whether or not associated injuries are present.

6. *The pulse* is usually rapid and thready in the case of a massive hemorrhage; otherwise, it may be normal in character.

7. *Temperature* is either normal or subnormal immediately following the injury; however, in a few hours it begins to rise and reaches mild febrile degrees.

8. *Percussion* of the abdomen reveals in many cases Pitts' and Balance's sign. This sign, described in 1896, is manifested by dullness in the left flank when the patient is turned on the right side and by sonority

of the right flank when the patient is turned on the left side. In other words, there is a dull region on the left flank which is established in a definite position. It consists of fixed clots of blood. Its presence is considered as pathognomonic for splenic rupture although its absence does not speak against rupture.

9. *Hemorrhage.* This is a frequent symptom. The extent, site and nature of the rupture determines the amount of hemorrhage. In the majority of cases hemorrhage is moderate. In case of massive hemorrhage the patient complains of vertigo and tinnitus aurium. The pulse is rapid and small, the respiration is superficial and the temperature is subnormal. The patient is pale; the extremities are cold and the skin is clammy and cold.

When there is severe hemorrhage, death occurs within a few hours. Berger states, basing his statement on material of 467 collected cases, that in all instances of death due to hemorrhage 52 per cent died within one hour, 14 per cent from one to sixteen hours and the remaining 34 per cent died from sixteen to twenty-four hours after injury. The reason why many patients survive the hemorrhage is the fact that the latter stops due to clotting, a drop in the blood pressure occurs and the extravasated blood acting as a tampon may encyst and compress the place of rupture.

The hemorrhage may be interstitial, filling the place between the parenchyma, or it may be subcapsular. These two conditions are possible only when the capsule is not ruptured. Even when the capsule is ruptured this does not mean that the blood will effuse into the free peritoneal cavity; in cases in which perisplenic adhesions were formed before the rupture, the blood may encyst there and compress the spleen.

Finally, hemorrhage may stop soon after the injury and appear hours or days later. This symptom is known as *delayed hemorrhage*. Pitts and Ballance collected in 1896 seventeen cases of delayed hemorrhage. The longest time on record for delayed hemorrhage was nineteen days. The period between the time of injury and of hemorrhage is known as the *latent period* (Baudet). Different explanations were given for this phenomenon of delayed hemorrhage. When a contusion of the spleen is present, in which the capsule is intact, the blood accumulates within the capsule and the spleen enlarges in size; the capsule becomes tense and finally ruptures and the blood extravasates into the free peritoneal cavity; the blood within the spleen, which acted as a tampon, releases the pressure on the ruptured splenic vessels and an additional flow of blood ensues.

If the capsule is ruptured at the time of injury, the omentum or a blood clot may plug the rent in the capsule and stop the hemorrhage. When the plug is dislodged, hemorrhage ensues.

Vomitus occurs from time to time but it is not a constant symptom.

DIAGNOSIS. There are no pathognomonic signs of injury of the spleen and for this reason the diagnosis is not easy, particularly immediately after the injury. Associated lesions may obscure the signs and symptoms of a ruptured spleen. However, if there is a history of trauma, particularly in the splenic region, and symptoms showing injury to an abdominal viscus and of internal hemorrhage, it is highly probable that the spleen is ruptured. The presence of Kehr's and Ballance's signs are highly suggestive of ruptured spleen, though their absence does not speak against it. All in all, the diagnosis is usually made correctly in about 80 per cent of all cases.

Among the conditions from which ruptured spleen should be differentiated are the following:

Contusion of the Abdominal Wall. It may give the same symptoms as splenic rupture. The points of differentiation are that the pulse rate, blood pressure and blood count remain normal in case of contusion.

Fracture of the Ribs on the Left Side. Very often this is associated with ruptured spleen. An abdominal tap for blood may clear up the diagnosis.

Perforated Peptic Ulcer. This is accompanied by more intense peritonitic phenomena and by frequent hematemesis.

Other conditions in which a differentiation should be made are: ruptured kidney, acute pancreatitis, pneumonia, ruptured ectopic pregnancy, acute cholecystitis, rupture of the liver and acute appendicitis.

COURSE OF THE INJURY. If the rupture is left to its own course, the mortality rate varies from 85 to 90 per cent. The prognosis is much better in children.

TREATMENT. Surgical treatment is the only one advisable, because the mortality rate in conservative management ranges between 85 and 90 per cent, while the mortality rate for operative treatment is approximately 30 per cent. The operations employed are splenectomy, tamponade and the suture of the spleen. The operation of choice is splenectomy.

Tamponade should be used only when splenectomy is contraindicated as, for instance, poor condition of the patient, inaccessibility of the spleen, adhesions between the spleen and diaphragm making the delivery of the spleen difficult, etc.

Splenorrhaphy may be employed only when there is a moderate degree of tear in the capsule with rather superficial penetration of the tear into the parenchyma. If, however, the injury of the parenchyma is deep, or if there are multiple injuries of the capsule and parenchyma, splenorrhaphy is contraindicated.

Preoperative management consists in the combating of shock. It is advisable to give a blood transfusion preoperatively. If no blood is available immediately, the intravenous injection of dextrose in Ringer's solution is an excellent procedure. Only after that should the operation be performed. It usually requires a few hours between the time the patient

is admitted to the hospital and the time of operation. The argument that there is no time for waiting, because the patient is bleeding, is not valid at least in 86 per cent, because 52 per cent of those who die from hemorrhage succumb within the first hour after the accident and 34 per cent die between sixteen and twenty-four hours, thus leaving only 14 per cent who die between one and sixteen hours after the accident, in which case the delay may be responsible for the fatal outcome.

SPONTANEOUS RUPTURE OF THE SPLEEN. Under this name are included those cases of rupture of the spleen without any visible cause such as a blow, fall, etc. They occur in pathologically changed and in overdistended spleens, which are to be found in malaria, relapsing fever, typhus fever, typhoid fever and in acute septic splenitis. However, cases are reported in which spontaneous rupture of a normal spleen has occurred. Nevertheless, spontaneous rupture of the spleen is a rarity. Melchior, in 1911, collected twelve cases of spontaneous rupture of the spleen in typhoid fever. Leighton, in 1921, collected only seventy-three cases of spontaneous rupture of a malarial spleen. C. Zuckerman and Jacobi, in 1937, collected from an exhaustive study twenty cases of spontaneous rupture of a normal spleen. This occurs usually by straining or when the patient lifts a heavy object. The symptoms are identical with those of a subcutaneous rupture of the spleen.

Open Wounds of the Spleen

Open wounds of the spleen are either of a gunshot or of a stabbing variety. The first type occurs in nearly 6 per cent of all wounds of the abdominal viscera and the second type in about 2 per cent. These injuries may be isolated or they may involve other structures in the abdominal or chest cavities or in both.

Due to the fact that in the case of inspiration the expanded lungs cover the upper two-thirds of the spleen and that the pleura covers either the entire or nearly the entire spleen, any injury to the spleen by an object, which entered from the chest wall, is bound to injure either the lung or the pleura. A great number of cases reported corroborate this point. Shaefer, in studying seventy-one cases of open wounds of the spleen, found only one instance in which it was of an isolated type. In the statistics collected by Finkelstein only six out of sixty-one instances of open injuries to the spleen were isolated cases. Among the associated injuries which are encountered most frequently are those of diaphragm and pleura, then of the stomach, liver and bowels. Because the stomach, splenic flexure of the transverse colon, left kidney, tail of the pancreas and diaphragm are situated close to the spleen, it is only natural to expect that injury to the spleen will often be associated with the injury to some of these structures.

The gravity of the situation and the prognosis in each individual case depend on whether the injury is isolated or combined.

SYMPTOMATOLOGY is variable, depending on the presence of associated lesions and their degree of injury. In cases of severe intraperitoneal hemorrhage from the spleen, the loss of blood is most conspicuous. In cases of injury to the gastrointestinal tract with a moderate degree of hemorrhage, the symptoms of a ruptured viscus or of peritonitis are preëminent. Among the symptoms most frequently met with are paleness of the patient, accelerated pulse, muscular rigidity, vomitus and occasionally dullness in the left hypochondrium. These symptoms indicate an abdominal catastrophe.

In a concomitant chest injury, hemopneumothorax will take place very often and the patient will become dyspneic, cyanotic and restless. These latter symptoms may even obscure the abdominal symptoms, particularly when the abdominal injury is confined to the spleen alone and is not accompanied by any severe form of hemorrhage.

TREATMENT. Open wounds of the spleen always require surgical intervention. The method of approach may be either transpleural or transperitoneal. The transperitoneal approach gives a better exposure of the abdominal cavity and, therefore, should be preferred to the transpleural approach. The operations employed are splenectomy, splenorrhaphy and tamponade. Splenorrhaphy may be employed in stab wounds of the spleen; however, splenectomy is preferable. In any case, the mortality in open wounds is appalling. The mortality rate for combined injuries is between 60 and 90 per cent and for isolated cases it is about 50 per cent for gunshot wounds and 20 per cent for stab wounds. The immediate cause of death is hemorrhage, heart failure and peritonitis.

TORSION OF THE PEDICLE

Not all displaced spleens require surgical treatment and fewer demand urgent surgical intervention. This is reserved for those cases only in which there is torsion of the splenic pedicle. If the spleen leaves its normal seat and descends downward, it is known as *ectopic spleen*; if it leaves its seat entirely in the left hypochondrium, it is known as *wandering spleen*.

ETIOLOGY. Up to the year 1938, 103 cases of torsion of the splenic pedicle were reported and nearly all of them were in females; only eleven were encountered in males. The first report of torsion of the pedicle in a male was made in 1918 by Petridis. John E. Sutton, Jr., in 1925, reported the first case of a male operated upon in the United States.

The underlying causes of splenoptosis are obscure. Some of the cases seem to be congenital and others acquired. The congenital factors are two: the length of the splenic pedicle and the conformation of the abdominal cavity. The latter consists of more shallow paravertebral niches so that it is easier for the spleen to prolapse. The fact that splenoptosis occurs most frequently in women leads to the belief that it may also be due to a relaxed abdominal wall. However, when we consider that

among the ninety odd cases more than twenty never bore children, that there are many cases in which general enteroptosis exists in which all the viscera but the spleen were prolapsed, and that there is another group of cases in which only the spleen and no other viscera were prolapsed, we come to the conclusion that ectopy of the spleen is not due only to relaxation of the abdominal wall. Some authors believe that there is not a single factor which produces ectopy of the spleen but a combination of factors, namely, congenitally elongated pedicle, tearing of the ligaments by trauma, traction of a diseased heavy spleen and relaxation of abdominal muscles. The pedicle may be of a great length; the longest described was ten inches. In about 67 per cent of all cases torsion occurred between the ages of twenty and forty, in 15 per cent before the age of twenty and in 18 per cent after the age of forty.

Torsion of the splenic pedicle occurs in about 29 per cent of all ectopic spleens. Johnston collected 708 cases of splenectomy; in 139 they were done for ectopic spleens, of which thirty-nine presented a twisted pedicle. This rotation may take place gradually or suddenly; it may turn around itself from one to four times. The blood circulation of the pedicle is partially or completely arrested and thrombosis of the splenic vein and its tributaries is common. The spleen becomes congested; very often infarction and hemorrhages, or gangrene ensue depending on the degree of vascular disturbances.

SYMPTOMATOLOGY. Torsion of the spleen occurs in one of three forms: acute, subacute and chronic. The symptoms will differ essentially depending on the type.

ACUTE TORSION. About 60 per cent of all cases are of the acute type; in one-third of these cases the symptoms will appear as from a clear sky; however, in two-thirds of the cases the patients will give a history of abdominal symptoms antedating the torsion and which may be ascribed to the splenic displacement. However, there are no symptoms pathognomonic of torsion of the pedicle; they are the symptoms of an acute condition of the abdomen, namely, pain, which is localized in the epigastrium or is generalized, nausea and vomitus, tenderness and rigidity. As the spleen very often is displaced into the left, or even into the right lower quadrant, the location and character of the pain and tenderness resemble those of a twisted pedicle of an ovarian cyst or of appendicitis.

The temperature is either normal or slightly elevated; the pulse is rapid and there is a moderate leukocytosis. The abdomen is moderately distended. However, in about 8 per cent of all recorded cases the spleen actually compressed the entire lumen of the bowel and produced intestinal obstruction. Palpation reveals a mass in any part of the abdomen and, as a matter of fact, the most frequent location is not in the left upper quadrant as one may be inclined to believe.

According to the statistics collected by Irvin Abell, out of eighty-five

cases the spleen was found in the pelvis and lower abdomen thirteen times, in the pelvis alone ten times, in the right lower quadrant nine times, in the left lower quadrant eight times, in the left upper quadrant seven times, in the epigastrium twice, etc.

When occupying a pelvo-abdominal or pelvic position, the spleen may be wedged between the urinary bladder and the uterus and produce symptoms of retroversion of the uterus and of urinary bladder irritation.

SUBACUTE TORSION. This type is characterized by repeated attacks of pain and tenderness of lesser severity, separated by periods without any symptoms. During the attack the spleen may be palpable.

CHRONIC TORSION. Chronic torsion takes place gradually. There is no complete strangulation of the blood vessels. The spleen becomes enlarged and tender by palpation. Accordingly, the symptoms develop gradually.

DIAGNOSIS. As there are no symptoms pathognomonic of torsion of the splenic pedicle, the diagnosis is very difficult and, with very few exceptions, no correct diagnosis has been made prior to operation. If there is a history of a wandering spleen, of attacks which previously took place, and if a mass is palpable which can be replaced into the left hypochondrium, or if a notch between the convex and the concave surfaces of the mass is discernible, indicating the presence of the spleen, a correct diagnosis can be made. In many instances a diagnosis has been made of a twisted ovarian cyst, kinked hydronephrosis, intestinal obstruction, perforated gastric or duodenal ulcer, peritonitis and tumor of the kidney. Some of the conditions can be differentiated from torsion of the pedicle such as perforated gastric or duodenal ulcer by the presence of air bubbles, as detected fluoroscopically, or tumor of the kidney by lack of mobility of the mass and the presence of urinary symptoms. However, in many cases the differential diagnosis is impossible. Out of ninety-seven cases of torsion of the splenic pedicle, as collected by Abell, only in nineteen was a correct preoperative diagnosis made. In the remainder of the cases other organs were blamed or the diagnosis of "acute abdomen" was made. The erroneous diagnosis of ovarian cyst was made most frequently, then in their order of frequency appendicitis, intestinal obstruction, peritonitis, perforated gastric or duodenal ulcer and floating kidney.

TREATMENT. Two procedures are available for torsion of the spleen. The operation of choice is splenectomy. Another procedure occasionally employed is splenopexy with preliminary detorsion of the splenic pedicle. Seven cases of detorsion alone were reported and in two of these cases recurrences took place. The mortality is high and depends on the complications present and the time of operation. In Johnston's series of thirty-six cases in which the patients were operated upon the mortality rate was 26 per cent.

Splenectomy is the better method because, due to torsion and to frequent thrombosis of the splenic vessels, the spleen is considerably damaged and there is no good reason for leaving it. The insecurity of the normal position of spleens which are inherently ectopic, and the fact that there are no known methods which will prevent torsion, speak against conservative methods. On the other hand, the ease with which other tissues rich in reticulo-endothelial cells compensate for the removed spleen invalidates any objection to the loss of its function.

ABSCESS OF THE SPLEEN

Abscess of the spleen is considered a rarity although some investigators are of the opinion that it occurs more frequently than some believe but that it is not recognized. Grand-Moursel, in 1885, collected fifty-seven cases, Küttner, in 1907, 116 cases, Wallace in 1922, more than 100 cases. A. E. Billings reports that out of 3,600 autopsies thirty cases of abscess of the spleen were found or an incidence of 0.4 per cent. It may be of primary or of metastatic origin. As a primary lesion it is very rare, much rarer than the metastatic type. If it is of the latter class, the abscess may come from any organ situated close to or at a distance from the spleen.

ETIOLOGY. Abscesses of the spleen usually have a secondary origin although occasionally they may have a primary origin. The source of infection in the case of metastatic origin may be in a neighboring organ or in a distant part of the body. Elting states that almost all pathogenic bacteria, except gonococci, were isolated from a splenic abscess. The *Bacillus typhosus*, streptococcus, staphylococcus aureus, *Bacillus coli communis* and pneumococcus are found most frequently. The infection usually enters through a blood stream or by virtue of its close relationship to a neighboring viscus, such as the stomach or the liver.

PATHOLOGY. All abscesses of embolic origin and a majority of those of traumatic origin are situated intracapsulary. For this reason the spleen is movable until the abscess reaches and then penetrates the capsule, at which time the spleen becomes adherent to the surrounding viscera or to the diaphragm. If the abscess is located in the upper pole and perforates, it forms a subdiaphragmatic abscess; if it is located in the lower pole and perforates, it produces general peritonitis. Occasionally, the splenic abscess perforates into the stomach, the intestine or through the diaphragm into the pleural cavity, or outward through the abdominal wall.

The abscesses are usually multiple although they may be single. In about 80 per cent of all cases the splenic abscess originates from an abscess in another viscus. It is extremely important to bear this fact in mind when selecting the proper cases for surgical interference as it has considerable bearing on the prognosis.

SYMPTOMATOLOGY. The symptoms may be acute, subacute or chronic. These vary greatly depending upon whether the abscess is of primary or of metastatic origin, whether it is situated deep in the splenic tissues or has broken through the capsule, whether it lies in the upper or in the lower pole and upon the character of the bacterial flora.

There are general symptoms of infection, such as elevated temperature, chills and sweats, accelerated pulse and leukocytosis, and localized symptoms, such as pain, tenderness and rigidity in the left hypochondrium. If the abscess is located centrally, far away from the capsule, the pain may be absent; if the abscess is situated in the upper pole and breaks through the capsule and irritates the diaphragm, pain will be present in the left shoulder (Kehr's sign). This sign has great diagnostic significance. In such a case the abscess may push the spleen downward and the left side of the diaphragm upward. This can be demonstrated by x-ray. In general, an abscess at the upper pole gives signs of thoracic irritation, whereas an abscess at the lower pole gives signs of abdominal irritation. Exploratory aspiration when positive is of the greatest importance, although a negative result does not rule out an abscess. However, I do not believe that this procedure should be carried out because it is fraught with danger. A needle, contaminated by the contents of the abscess, may infect the pleural cavity if the needle made its way to the abdomen through the pleural cavity.

Diagnosis is a difficult one. One has to differentiate from other conditions which are manifested by splenic enlargement and give at the same time general systemic reaction indicating the presence of a general infection. Differentiation must also be made from other conditions, such as abscess of the liver, subphrenic abscess, nephritic abscess and empyema.

TREATMENT. The mortality in surgically treated patients depends on many factors: Whether the spleen is movable or adherent, the general condition of the patient, the type of infection which produced the abscess, and so on. For all cases, the surgical mortality is about 20 per cent, ranging from 9 to 38 per cent, according to different authors, whereas the mortality for medically treated patients before the advent of sulfa drugs and penicillin has been about 80 per cent. Undoubtedly penicillin and sulfa drugs will lower the mortality.

If the abscess is of pyemic origin and the patient shows grave symptoms due to the original disease, surgical interference will be accompanied by a very high mortality. In other words, only those patients should be operated upon who are fairly good surgical risks. The operations employed are splenotomy and splenectomy.

Splenotomy in this particular type of case is actual drainage of the abscess. It is easier to perform than splenectomy, although the mortality rate is about as high as that in cases of splenectomy. In case of splenotomy the approach may be:

(a) **Abdominal.** This provides a good approach. The only objection is the possibility of infection of the general peritoneal cavity.

(b) **Transpleural—transdiaphragmatic route.** This approach may be carried out for such cases in which there is a subdiaphragmatic abscess. The operation is performed in two stages. The advantage in this method is the excellent approach to the upper pole and to the subdiaphragmatic space. The weak feature of this method is the possibility of contaminating the pleural cavity.

(c) **Posterior approach** through the diaphragm below the pleural sinus is possible only for exposure of subdiaphragmatic abscess but not for splenectomy.

The operative technic of these procedures is described below.

CYSTS OF THE SPLEEN

Cysts of the spleen are rather infrequent. They are far less frequent than those occurring in the ovary, kidney or liver. These cysts are either parasitic—echinococcal cysts—or nonparasitic. The parasitic cysts occur twice as frequently as nonparasitic cysts. The latter, up to January, 1939, numbered only 137 reported cases.

Nonparasitic cysts. Nonparasitic cysts include a large number with varied contents, size, cell lining and mode of origin. They have been designated as true and false: the former have a cellular lining; false cysts occur four times as frequently as true cysts.

From the standpoint of contents they are subdivided into hemorrhagic and serous or lymphatic. The hemorrhagic type occurs nearly two and one-half times as frequently as lymphatic cysts.

Cysts occur more frequently in females (3:2) and nearly 75 per cent occur between the ages of 10 and 50, whereas only 10 per cent occur under the age of 10 and 15 per cent above the age of 50.

As antecedent diseases, trauma was present in 23 per cent and malaria in 74 per cent. In most of the cases cysts are solitary.

SYMPTOMATOLOGY. As there are no apparent functions of an adult spleen, there are no symptoms which are the direct result of the involvement of the splenic tissue. The appearance of symptoms depends on the size of the cyst. If the cyst is large, symptoms of pressure appear. These symptoms vary depending on the structure that it compresses. In addition, direct results of pressure may contribute to formation of adhesion, which in their turn may produce new symptoms. If symptoms are present the chief one is pain. It is either a dragging or a feeling of heaviness and is located either in the left hypochondrium or in the epigastrium. However, in some cases in which the spleen is displaced the pain may be felt in the region where the bulk of the spleen is located.

Digestive disturbances may appear in the case of abdominal pressure; obstructive symptoms may be present in the case of pressure on the

bowel, and dyspnea, if the pressure is exerted on the diaphragm. The same pressure on the diaphragm produces circulatory disturbances.

Inspection may reveal a swelling of the abdomen, either in the left hypochondrium or in any other portion. Palpation may reveal a tumor extending from the left costal arch medialward to the midline or even beyond, to the right and below to the navel or beneath it, depending on the size of the cyst. This mass may be either movable or fixed. Fluctuation may be present but quite often it is absent. Ascites usually is not present.

The DIAGNOSIS depends upon the physical examination and roentgenologic evidence. That the diagnosis is difficult may be attested by the fact that a correct diagnosis is seldom made. If a tumor is palpated in the left hypochondrium and is located rather superficially, a tumor of the spleen should be suspected; if the tumor lies deeper, one may suspect a tumor of the left kidney. If a notch is felt, it is definite that the viscus is the spleen. If the tumor fluctuates, this reveals the cystic nature of the tumor. The cyst of the spleen also should be differentiated from those of the omentum, pancreas, mesentery and left lobe of the liver. If the spleen is misplaced into the pelvis, the spleen may be mistaken for an ovarian cyst or floating kidney. X-ray diagnosis is of great diagnostic aid. Benton called attention to the fact that the x-ray shows the displacement of the lower third of the esophagus and the stomach to the right and the downward displacement of the splenic flexure of the colon. He was the first to show that these organs come to their normal position after splenectomy has been performed. Benton considers this downward displacement of the splenic flexure of the colon as almost pathognomonic for large cysts of the spleen. In addition to the picture shown by Benton, one can see on the x-ray film that in cases of large tumors the diaphragm is elevated on the left side by the growth, the left lower ribs bulge, the psoas muscle shadow is obliterated and frequently there is a downward displacement of the left kidney. Ostro and Makover noted that if the splenic enlargement is due to myeloid leukemia, elevation of the diaphragm, spreading of the ribs and obliteration of the psoas shadow do not occur. Their interpretation is that in case of splenic myeloid leukemia the growth of the spleen is downward and forward and the growth in this direction does not produce the above enumerated pressure effects.

After the diagnosis of cyst has been made, one has to decide whether it is parasitic or not. In the case of a *parasitic* cyst it is echinococcal. The diagnosis can be made before the operation, by employing the Casoni reaction or Weinberg reaction, or at the time of operation when, after the abdomen is opened, the echinococcal cyst is differentiated from a non-parasitic cyst by its characteristic appearance: It is usually elongated or pointed and springs from the lower pole.

TREATMENT. Methods of treatment are the following: (a) Splenectomy (b) excision of the cyst and (c) incision and drainage.

Splenectomy is the operation of choice; however, its execution is often precluded because of the strong adhesions present.

Incision and drainage may be done either as a one-stage or as a two-stage operation. In the latter, the first stage is confined to laparotomy and to bringing the spleen to the abdominal wall so as to produce adhesions between the parietal peritoneum and the spleen beyond the cyst so that the portion of the spleen with the cyst becomes extraperitonized. The second stage consists in the opening and draining of the cyst by a gauze roll and the ends of the tampon are brought outside. However, in case of an echinococcus cyst one should be extremely careful not to contaminate the peritoneal cavity by the contents of the cyst. For this reason it is advisable, in addition to walling off the cyst from the rest of peritoneal cavity and to aspirating the contents of the cyst, to inject into the cyst (after it has been emptied by aspiration) 300 to 400 cc. of formol (depending on the size of the cyst) and to leave it there for three to five minutes in order to kill the scolices.

Excision or enucleation of the cyst was recorded only in a few cases. This procedure is fraught with the danger of hemorrhage. In the few successfully performed cases the surgeons placed a series of interrupted sutures around the cyst before it was excised. However, we do not advise this method. Multiple cysts, lymphangiectasis and polycystic degeneration should be treated by splenectomy as the first choice.

The mortality in cases reported proved to be lower when splenectomies were performed.

Operative Technic

The spleen is exposed by one of several incisions, depending on whether the approach is abdominal, transpleural, subpleural or transperitoneal. In case of a transperitoneal abdominal approach, the following incisions are used.

(a) **Left midrectus** (Fig. 37, B). This incision is carried along the middle of the left musculus rectus abdominis, starting from the costal arch and reaching the level of the umbilicus.

(b) **Left midrectus with addition of a transverse incision.** The vertical portion of the incision is carried out as in the previous incision and the transverse incision is done perpendicularly to the first one at the level of the cartilage of the ninth rib.

(c) **Left subcostal** (Fig. 37, A). This incision runs parallel to the left costal arch starting at the point of junction of the seventh costal cartilage with the sternum and runs until it reaches slightly lateral to the left border of the left musculus rectus.

The incisions (a) and (b), are sufficient for splenotomy and splenopexy and in some cases for splenectomy. The incision, (c), should be used for those cases of splenectomy in which the spleen lies high and deep and is not easily accessible.

In case of a **transpleural approach**, which may be used only for drainage of a splenic abscess, the incision is made in the following manner:

Stage I: Step 1. The skin incision starts along the eighth or ninth rib from the posterior axillary line and runs forward until the cartilage of this rib is reached.

Step 2. The eighth and ninth ribs are resected subperiosteally for a distance of four inches each and the pleural sinus is exposed at the level of the eighth rib.

Step 3. The costal pleura is sutured to the diaphragmatic pleura in a circular manner.

Stage II. This is done several days later.

Step 1. The skin is reopened.

Step 2. The periosteum of the ninth rib, the endothoracic fascia, the costal pleura, the diaphragmatic pleura, the fascia covering the thoracic surface of the diaphragm, the muscular part of the diaphragm, the abdominal side of the diaphragmatic fascia and the parietal peritoneum covering the diaphragm are opened and the abdomen is entered. Thus, the opening is made *below* the suture line which united the costal to the diaphragmatic pleura in the first stage of the operation.

This approach was first described and used by Trendelenburg in 1883. However, it may be used only when the pleura is already involved; otherwise, if one decides to use the transthoracic approach, it should be subpleural. The approach may be either anterior or posterior. For access to the spleen the anterior approach is used more frequently. It was introduced by Lannelongue in 1887 and modified by Canniot in 1891. The incision is carried out in the following manner:

Step 1. The skin incision starts 3 cm. lateral to the ensiform process and 2 cm. below the costal arch. It is carried out laterally as far as the bony portion of the tenth rib, which corresponds on the surface marking to the middle axillary line. The skin and superficial and deep fascia are incised; the musculus rectus, obliquus abdominis externus and internus are cut, and the costal arch is raised. The attachment of the transversus abdominis muscle and the diaphragmatic muscle are carefully detached from the cartilages of the arch. **Do not separate** the fibers of these muscles from the bony portions of the ribs, since the pleura and the pericardium may be opened.

Step 2. The cartilages of the eighth and ninth ribs are resected. This will expose a space in which will be seen the fibers of the internal oblique muscle, transversus abdominis muscle and the diaphragm.

Step 3. The internal oblique and the transversus abdominis muscles are divided. The pleura is now displaced upward, the diaphragm with the peritoneum are divided and the abdominal cavity is entered.

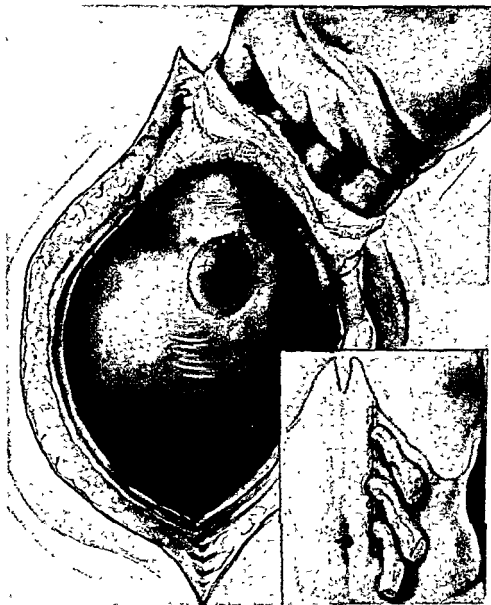


FIG. 155 Splenotomy for a cyst.

Splenotomy (Fig. 155).

The term consists of two Greek words *σπλήν* spleen, and *τέμνω* to cut. It signifies an incision of the spleen.

Indications. This operation is done either for evacuating an abscess

or a large single cyst. If abscesses are multiple or if numerous cysts are present, splenectomy is indicated. The operation may be done either as a one-stage or as a two-stage operation. In the case of a one-stage operation the technic is as follows:

Step 1. The abdomen is opened by one of the incisions mentioned above.

Step 2. The spleen is exposed and "walled off" from the rest of the peritoneal cavity by two rolls of gauze, one end of which leaves the abdominal cavity through the upper end and the other through the lower end of the abdominal wound. Exploratory puncture by a needle is made to ascertain the exact location of the abscess. If pus is found, an incision through the capsule is made and the pus is evacuated. A roll of gauze is introduced into the abscess cavity and the end of the gauze is brought outside the abdominal cavity. The abdomen is closed in layers; an opening is left at the upper and lower angles of the wound for the ends of the gauze rolls.

Three or four days later the two rolls of gauze which "walled off" the spleen from the rest of the peritoneal cavity are removed. A few days after removal of the "walling off" gauze the roll of gauze which was inserted into the abscess cavity is removed.

Splenorrhaphy (Fig. 156)

This method consists of suturing the spleen and is applicable only when there is but one wound present which is not large. If the wounds are multiple or if it is difficult to stop the bleeding, splenectomy should be performed.

TECHNIC.

Step 1. The abdomen is opened and the spleen is exposed.

Step 2. A large piece of omentum is excised and tucked snugly into the open wound of the spleen. This will act not only as a tampon but as an active coagulant. The edges of the capsule of the spleen are sutured to each other with heavy catgut over the tucked piece of omentum (Fig. 156).

Splenopexy

This operation consists in the fixation of the spleen to the anterior abdominal wall. It is done in some cases of a movable spleen in which there is no torsion of the pedicle, and it may be done occasionally in cases of torsion of the pedicle in which the blood vessels are not thrombosed. However, in cases of torsion of the pedicle, whenever possible, splenectomy should be preferred to any other procedure. Among other conditions for which this operation is indicated we may mention ascites due to cirrhosis of the liver. The fixation may be either intraperitoneal or extraperitoneal or combined intra-and-extraperitoneal.

The first splenopexy was performed by Tuffier in 1882. Since that time several methods have been employed.

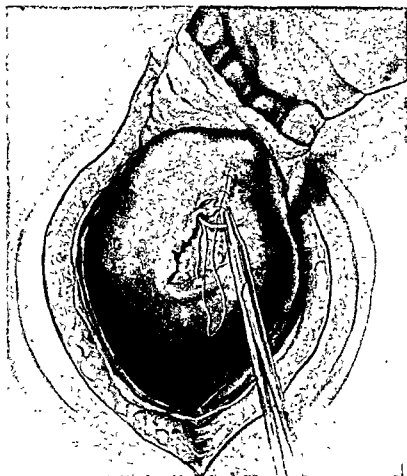


FIG. 156. Splenorraphy.

Kouwer Method

This method consists of fixation of the spleen to the diaphragm by forming adhesions between these two structures. In order to accomplish it, Kouwer placed rolls of gauze between the upper and lower poles of the spleen and the diaphragm; the ends of the rolls are brought outside the abdominal cavity. The spleen is allowed to drop back into its normal position; thus, adhesions are formed between the concave surface of the spleen and the diaphragm. In three or four days the rolls of gauze are removed. Some surgeons rub the convex surface of the spleen and the concave surface of the diaphragm before placing the rolls of gauze.

Rydygier Method

Step 1. The abdomen is opened by a longitudinal incision in the left hypochondrium (Fig. 157, A).

Step 2. A slightly convex incision running lateralward and upward (Fig. 157, B), the length of which is equal to the width of the spleen, is



FIG. 157. Splenopexy (Rydygier method). A. Abdominal incision.
B. Incision of parietal peritoneum.

made on the inner surface of the thoraco-abdominal wall extending from the eleventh to the ninth ribs. The peritoneum of the lower lip is separated by the finger from the superimposed soft tissues, so that a pocket is formed with its bottom directed downward (Fig. 158 C). The lower pole of the spleen is placed in this pocket. In order to prevent further separation of the peritoneum from the superimposed structures of the pocket by the lower pole of the spleen, the peritoneum is sutured to the

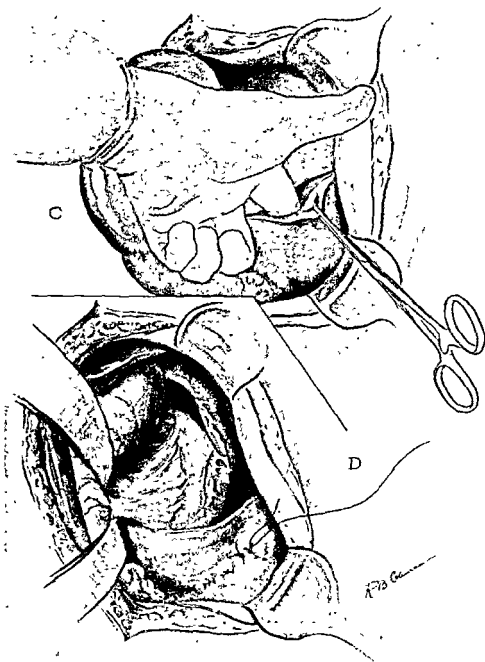


FIG 153. Splenopexy (Rydygier method). C Blunt dissection of parietal peritoneum from the lateral abdominal wall making an extraperitoneal pocket. D. Placing the lower pole of the spleen into extraperitoneal pocket. A series of interrupted sutures joining the peritoneum to the adjacent structures just below the lower pole prevents separation of the parietal peritoneum in a downward direction.

superficial structures by a series of interrupted sutures, approximately half an inch away from the distal end of the bottom of the pocket (Fig. 158, D).

Step 3. The upper lip of the peritoneum is undermined from the muscle of the diaphragm so as to form an upper pocket into which the upper pole

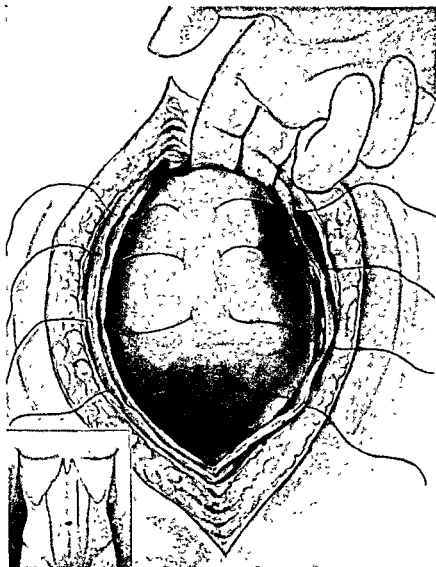


FIG. 159. Splenopexy (Tuffier method).

of the spleen is placed. Step 3 is done only if it is mechanically feasible. The characteristic feature of Rydygier's method is step 2.

Tuffier Method of Splenopexy (Fig. 159)

This is the oldest method of splenopexy and was reported in 1882. It consists in suturing the spleen to the abdominal parieties.

Step 1. The abdomen is opened by one of the abdominal incisions mentioned above and the spleen exposed.

Step 2. A bite is taken through the entire thickness of the abdominal wall, except the skin, of one lip; the needle then punctures the spleen penetrating not only the capsule but also some portion of the parenchyma and then the entire thickness of the abdominal wall, except the skin, of the other lip. The number of sutures depends upon the size of the spleen. These sutures are not tied at this time.

Step 3. All thickness of the abdominal wall except the skin are sutured in layers.

Step 4. The ends of the splenopexy sutures are tied.

Step 5. The skin is closed.

Bardenheuer Method of Splenopexy

This method, as introduced by Bardenheuer, was described by one of his associates, Plücker, in 1895. This procedure is characterized by

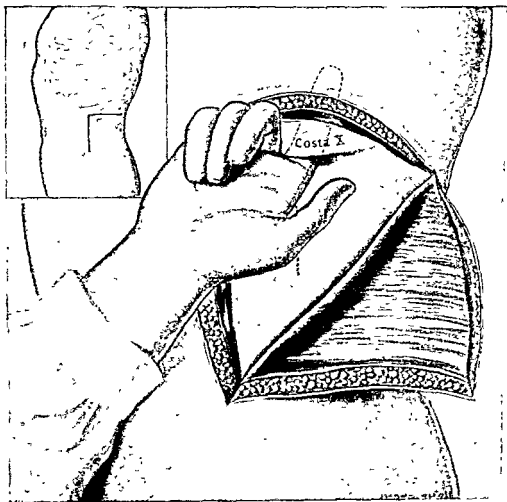


FIG. 160. Splenopexy by Bardenheuer method. (From Julius L. Spivack's "The Surgical Technic of Abdominal Operations," 4th edition 1946. Courtesy of Charles C Thomas, Publisher, Springfield, Illinois.)

placing the spleen extraperitoneally, attaching the hilum to the peritoneum and fixing the lower pole to the tenth rib.

Step 1. The abdomen is opened by an incision, starting at the left mid-axillary line just above the crest of the ileum and extending in an upward direction for four inches until the tenth left rib is almost reached.

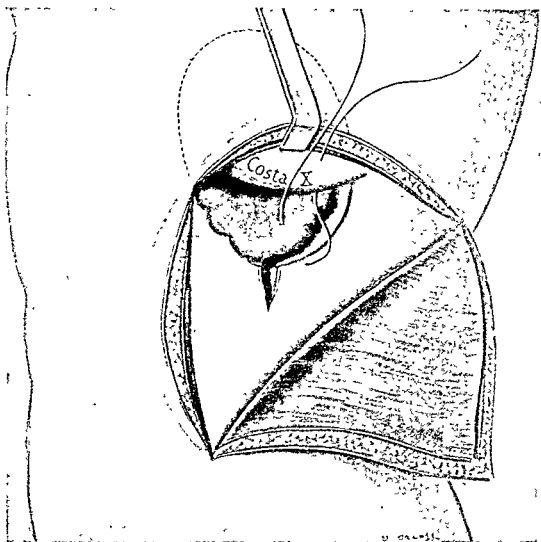


FIG. 161. Splenopexy by Bardenheuer method. (From Julius L. Spivack's "The Surgical Technic of Abdominal Operations," 4th edition 1946. Courtesy of Charles C Thomas, Publisher, Springfield, Illinois.)

Another incision is made starting at the upper end of the previous incision and running laterally perpendicular to the first one. The length of the second incision is also four inches. The entire thickness of the flap thus formed is cut down to the peritoneum, which is not yet opened. This musculocutaneous flap is reflected (Fig. 160).

Step 2. The exposed peritoneum is separated from the superimposed soft tissues as far as possible upward and downward (Fig. 160). A small

incision is made through the peritoneum, just large enough to draw the spleen outside of the abdominal cavity (Fig. 161). The spleen is placed extraperitoneally in the sac thus formed. The slit in the peritoneum is sutured to the gastrosplenic ligament.

Step 3. A silkworm-gut thread is passed around the tenth rib (which is freely exposed for this purpose) and then through the lower pole of the spleen (Fig. 161). The ends of the thread are not tied for the time being. A series of interrupted sutures are inserted between the lowermost part of the separated peritoneum and the superimposed structures, so as to prevent separation of the peritoneum from these structures in a downward direction, thereby preventing a downward slipping of the lower pole of the spleen. The long ends of the silkworm-gut thread, which fixed the lower pole of the spleen to the tenth rib, are now tied and the ends are cut short.

Schiassi Method of Splenopexy (Splenocleisis)

This method consists of producing extensive adhesions between the spleen and surrounding structures, including the anterior abdominal wall. By thus fixing the spleen to the anterior abdominal wall the method becomes that of splenopexy, which prevents reappearance of a movable spleen with all the dangers which may come from it. However, this method is more than a mere splenopexy. A thick capsule is formed around the spleen which compresses that organ, thus diminishing the amount of blood passing through the spleen and increasing the degree of anastomosis of the veins between the portal and systemic venous systems. This method can be used as a substitute for splenectomy in cases in which the latter is hazardous. It is, indeed, the substitution of a physiological for a mechanical splenectomy.

Step 1. The abdomen is opened by a left subcostal incision starting at the midline and running parallel to and two fingers below the left costal arch until the left midaxillary line is reached.

Step 2. A roll of gauze is placed behind and below the lower pole of the spleen, another medial to the hilum, a third lateral to the lateral border, a fourth above and behind the upper pole, a fifth and sixth between the anterolateral and anteromedial surfaces of the spleen and the anterior abdominal wall. The ends of these rolls are brought outside the abdominal wound through the upper and lower angles. The anterior surface of the spleen is scarified.

Step 3. The abdominal wall is closed in layers.

On the fifth day the roll above the upper pole is removed and at each following day another roll of gauze is removed. Finally, the last roll is removed from the lower pole.

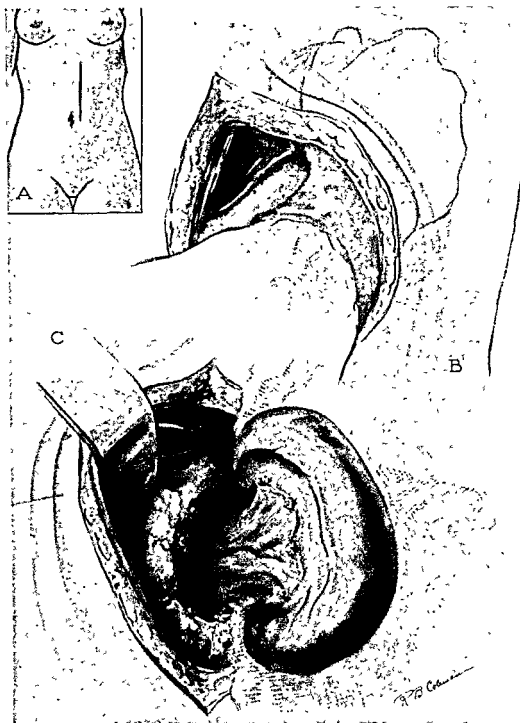


FIG. 162. Splenectomy. A. Abdominal incision. B. Mobilization of the spleen with the right hand preliminary to lifting it from the splenic bed. C. Gastrosplenic ligament cut between ligatures.

Splenectomy

This operation is performed for different conditions. In urgent surgery it is done in case of rupture of the spleen, torsion of the pedicle in case of wandering spleen, in some cases of abscess of the spleen and at times for cysts of the spleen. This operation, from the technical point of view, may be very simple when the pedicle is long, the spleen movable and no adhesions are present. However, in cases in which extensive adhesions are present, the operation may offer insurmountable difficulties.

There are two methods used in removing the spleen, namely, either to start with separation of the spleen from the diaphragm, with ligation of the pedicle as the last step of the operation, or to start the operation with ligation of the pedicle first, and separation of the spleen from the diaphragm as the last step.

Each operative step has its advantages and disadvantages depending on the circumstances. In most instances it is more advantageous first to separate the spleen from the diaphragm, deliver it outside of the abdomen as much as possible and then only ligate the pedicle.

Technic. Step 1. The abdomen is opened by one of the incisions previously described.

Step 2. The right hand is introduced into the abdominal cavity, placed between the spleen and diaphragm and the spleen is lifted from the diaphragm (Fig. 162, B). Immediately after the spleen is lifted, large packs of moist gauze are placed on the exposed bed of the spleen, thus checking the oozing from the diaphragm.

Step 3. The gastrosplenic ligament above the pedicle is divided between clamps, care being taken not to injure the fundus of the stomach which lies very close to the spleen (Fig. 162, C).

Step 4. Ligation of the pedicle may be done either "en masse" or in sections. The latter is more difficult. For less experienced surgeons ligation "en masse" is less dangerous. In this method three large curved artery forceps clamp the pedicle (Fig. 163, E), which is then cut between the clamp closest to the spleen and the middle clamp. In this manner, two clamps safeguard against hemorrhage from the splenic artery and one from the splenic vein. The stump is ligated twice with a double strand of No. 2 chromic catgut.

Before clamping the splenic pedicle one should obtain a clear view of it posteriorly, since not infrequently the tail of the pancreas reaches nearly to the hilum of the spleen and may be adherent to the splenic pedicle. By clamping the pedicle without previous separation from the tail, part of the pancreas may be caught in the clamp.

Step 5. All additional ligaments connecting the spleen with the surrounding structures should be cut between two clamps and carefully ligated.

Step 6. The moist pack is removed from the splenic bed of the diaphragm.

Step 7. The abdomen is closed in layers without drainage.

In cases in which the splenectomy is done by ligating the splenic

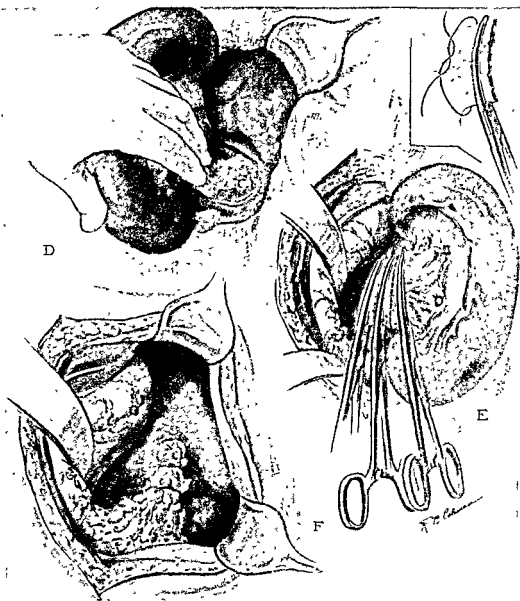


FIG. 163. Splenectomy. D. Pedicle of the spleen dissected away from the pancreas. E. Pedicle clamped, cut and ligated "en masse." F. Omentum is sutured over the tied pedicle.

pedicle first, some surgeons advise the ligation of the splenic artery first so as to give the vein a short time to carry away the blood from the spleen. One should remember that in the pedicle the splenic artery runs either as a single trunk or is divided into two branches. The vein is still

composed of two or three branches and they run below the arterial branches. Due to this arrangement (two arterial branches, several venous tributaries), the ligation of individual branches is not a simple procedure, particularly so when the exposure is not good. For this reason we advise the ligation of the stump "en masse." The technic then is as follows:

Step 1. The abdomen is opened.

Step 2. The spleen is pushed laterally, so as to expose the gastrosplenic ligament above the pedicle. The stomach is grasped and pulled medialward, which exposes the gastrosplenic ligament still more, and this ligament is divided between two clamps.

Step 3. The finger is introduced through the opening formed in Step 2 between the spleen and the stomach and slipped behind the pedicle. The finger should follow the pedicle closely; in this manner the pedicle will lie in front of the finger and the tail of the pancreas behind it. The pedicle is ligated either sectionally or "en masse."

Step 4. The spleen is separated from the diaphragm and removed.

Step 5. The abdomen is closed in layers.

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Chapter XII

Appendicitis

By JULIUS L. SPIVACK

Acute appendicitis is the abdominal condition that the surgeon is confronted with most frequently and comprises about one-third to one-half of all acute abdominal conditions. Its occurrence varies in different countries. Thus, in Sweden it occurs in 3 out of 1000 persons, in Germany in $3\frac{1}{2}$ out of 1000 persons, in the United States in $5\frac{1}{2}$ out of 1000 persons. One can have a clear conception of the frequency of this disease by stating that every minute one operation for acute appendicitis is performed in the United States. The mortality of acute appendicitis is very high and in spite of improved diagnosis and technic the mortality rate is not decreasing. In the United States in 1935, 16,142 persons died from appendicitis and it was the eleventh leading cause of death. In 1936, in the United States 16,480 persons died or 12.8 per 100,000. In this country one man dies from appendicitis every twenty-nine minutes.

SURGICAL ANATOMY. The point of origin of the appendix from the cecum is not constant. It may be found at almost any point of the cecal pouch. However, it is found most frequently at two points: (a) on the posterior cecal wall, slightly below the ileocecal junction, and (b) on the anterior cecal wall at its lowermost portion. The first position is found in about 90 per cent of the cases. The appendix may lie in front and medial to the cecum; it may lie below or retroceally. When it is retrocecal, the appendix may lie behind the parietal peritoneum or intraperitoneally. In exceptionally rare cases, it lies plastered against the posterior cecal wall behind the visceral peritoneum. It may also lie close to the psoas or to the iliacus muscle, against the right ureter, iliac vessels or pelvic wall.

POSITION OF THE APPENDIX (Fig. 164). The most frequent position of the appendix in an adult is behind the mesial portion of the cecum and it is often partially covered by the terminal portion of the ileum.

The second most frequent position is that in which the appendix lies lateral to the cecum and ascending colon, and the third position is one in which the appendix is in the pelvis. To these normally situated positions of the appendix may be added such in which it lies in abnormal positions. An appendix has been found to reach the front of the sacrum, the left pelvic wall and the left psoas muscle, resting on the lumbar vertebrae. The appendix together with the cecum has been found in the

left iliac fossa not as a case of situs inversus but due to an unusually long mesocecum and mesocolon which shifted the cecum with the appendix to the left side and became fixed there. The appendix has also been found in inguinal, femoral and umbilical herniae, in the foramen of Winslow and on the anterior stomach wall. All these conditions are due to the mobility of the cecum or of the colon ascendens. There are cases

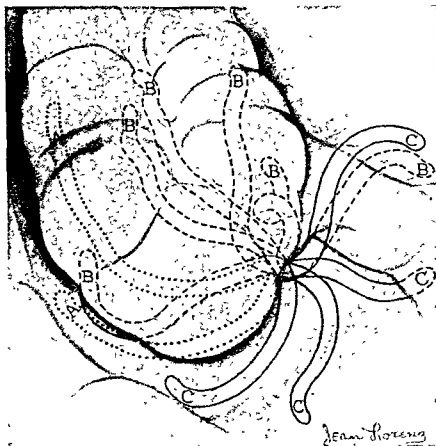


FIG. 164. Various positions of the appendix. A. Positions of the appendix behind the cecum and parietal peritoneum. B. Positions of the appendix behind the cecum but in front of parietal peritoneum. C. Positions of the appendix lying free in the peritoneal cavity.

in which the appendix occupies unusual positions due to arrested fetal development: the appendix is found in the splenic region and under the liver at the right hepatic flexure of the colon. In addition to these cases the appendix may lie in the left iliac fossa as a feature of situs inversus.

SIZE OF THE APPENDIX. The size of the appendix varies. The average size is about 9 cm. ($3\frac{1}{2}$ inches) long; however, cases have been described as small as 2 mm. ($\frac{1}{13}$ of an inch) and as large as 33 cm. ($12\frac{7}{8}$ inches). The lumen of the appendix averages 6 mm. and the thickness of its wall averages from 1 to $2\frac{1}{2}$ mm.

cases appendicitis may be due not to an infectious origin but to vascular occlusion and the inflammation appears as a secondary feature. From the morphologic picture, acute appendicitis may be subdivided into the following groups:

(A) CATARRHAL. In this type only the portion of the mucosa is affected. It may subside very soon or may spread into the deeper structures of the appendiceal wall and develop into one of the other forms.

(B) PHLEGMONOUS. In this kind of acute appendicitis the inflammation proceeds to other coats including the serosa. The appendix looks edematous and the tip of it is frequently slightly clubbed. It is tense and hyperemic; the blood vessels are well injected. Its color is bright red or covered with deposits of suppurated or necrotic tissue. The lumen of the appendix contains mucopurulent exudate.

(C) GANGRENOUS. This type generally occurs when appendiceal blood vessels are involved. If the artery is involved, anemic infarction follows; if the vein is involved, hemorrhagic infarct follows; these infarcts usually affect the antimesenteric border. These infarct areas become invaded by bacteria, which leads to suppuration and gangrene.

(D) PERFORATIVE. Perforation may take place in any type of appendicitis. The rupture may be of a pin-hole or of a larger size. A frequent cause of rupture is necrosis of the tissue by a concretion within the lumen of the appendix.

Symptomatology

This disease, as a rule, has no prodromal symptoms although in some cases the patient is constipated two or three days before the onset. In a typical case the disease comes as from a clear sky; there appears a number of symptoms in a definite succession which present definite characteristics. Not all of the symptoms are of equal importance; not all of them appear all the time and there is not a single symptom which is pathognomonic.

These symptoms in the order of their frequency and importance are: pain, nausea and vomiting, tenderness, rigidity, fever and leukocytosis.

PAIN. The significance of pain is of great importance. The correct interpretation of its character, severity, its variations, duration and localization may have a great diagnostic and prognostic significance. It is the earliest and the most constant symptom. It starts usually in the epigastric or in the umbilical region as a colicky pain. It stays in this region from a few to twenty-four hours and then localizes in the right lower quadrant. By this time the character of the pain changes. Its "colicky" character gives way to a steady, aching pain.

The feature of the pain, starting in the epigastrium and then localizing only in the right lower quadrant, is so characteristic for acute appendicitis, that pain starting in this manner is considered to be due to

appendicitis until proven otherwise, whereas pain starting at the right lower quadrant is not due to appendicitis unless proven otherwise. However, in rare cases the same character of distribution of pain is found in other acute intra-abdominal lesions, such as acute cholecystitis, salpingitis, perforated gastric ulcer or enterocolitis. On the other hand, in a definite number of cases, particularly in children and in elderly people, the pain may start in the right lower quadrant.

The severity of pain very often runs parallel with the severity and the extent of the appendiceal inflammation, particularly after the third day. However, *in the first two days the gross pathology is graver than the clinical manifestations.* Cases are known of a sloughed appendix with very slight pain. The individual temperament of the patient is also responsible for a different response to a seemingly identical degree of pathology. For this reason the severity of pain cannot serve as an absolutely reliable symptom.

Much more important than severity of pain is its duration, because it is indicative of the progress of the disease. In mild cases, when no complications arise, the pain subsides in twenty-four hours and is practically gone in forty-eight hours. If the pain continues and particularly if it increases in severity and in the size of the area involved after forty-eight hours, it is a sign of the spreading of the inflammation from the appendix to peritoneum. When the pain subsides slowly without cessation of other symptoms one should suspect gangrene of the appendix, and if it subsides suddenly, especially following a marked increase of its intensity and without cessation of other symptoms, perforation should be suspected.

NAUSEA AND VOMITUS occur very often. Nausea is nearly a constant symptom (85 per cent); vomitus is less frequent (42 to 50 per cent). Vomitus usually appears approximately three hours after the onset of pain; the patient vomits once or twice; the contents are at first of the gastric contents and the repeated vomiting consists of bile. When pain leaves the epigastric region and settles in the right lower quadrant, the vomitus ceases. This sequence of symptoms, namely, vomitus following pain is quite characteristic for acute appendicitis only, and for no other abdominal lesion. This means that if vomitus precedes pain in an acute condition of the abdomen, the case is not one caused by appendicitis. It should, however, be remembered that occasionally this is not the case. If the vomitus ceases after the pain is localized in the right lower quadrant and later reappears, it signifies that the inflammation has spread beyond the appendix and has involved the peritoneum.

TENDERNESS is a very important symptom. In its importance it is only second to pain. The correct interpretation of tenderness enables one to decide the seat of pathology, the degree of its development and the extent of its spreading. At first it is observed as a superficial, that

is as a cutaneous hyperesthesia that spreads all over the abdomen. It corresponds at this stage to the generalized abdominal pain. This tenderness is elicited by pinching or rubbing the skin between the fingers. After a few hours it gives way to "deep" tenderness, which is elicited by pressure at *McBurney's* or at some other point in the right lower quadrant. The deep tenderness and the localization of pain in the right lower quadrant usually appear at the same time; however, tenderness often appears before the pain is localized in the right lower quadrant.

The tenderness increases in strength and in twenty-four hours reaches its height. In moderate cases it fades away in forty-eight hours, in mild cases in twenty-four hours. It disappears gradually, never as abruptly as pain. If tenderness reappears, it is a sign of secondary peritoneal involvement.

Tenderness is more pronounced close to the seat of the pathology, when the appendix is more inflamed, when the seat of the pathology is near to the anterior abdominal wall and when the latter is very thin. Finally, the response of different individuals to pressure on the abdominal wall varies. Therefore, we can say that the degree of tenderness as observed in each individual is due to several factors: to the degree of pathology, its proximity to the anterior (or posterior) abdominal wall and to the perceptive mechanism of each individual patient.

It is usually considered that the point of greatest tenderness is at the base of the appendix. Here the afferent nerves of the appendix unite and carry the sensations to the spinal ganglia. This anatomical point being frequently constant at the cecum does not always correspond to the same point of the anterior abdominal wall on account of the mobility of the cecum. That is the main reason why several different points of greatest tenderness are described. Among the points most frequently mentioned are the following:

McBurney point is situated one and a half to two inches from the anterior superior iliac spine on a line drawn between the umbilicus and spina iliaca anterior superior (spino-umbilical line).

Morris point is on the spino-umbilical line at the point where the lateral border of the right musculus rectus crosses this line.

Lanz point is on the spino-umbilical line at the point of junction of its lateral and middle thirds.

Clado point is on the interspinous line (the line connecting the right and the left spina iliaca anterior superior) at the point where it crosses the lateral border of the right rectus muscle. However, the peritoneum close to the base of the appendix is early involved and becomes sensitive. For this reason rather a wide area is sensitive instead of a small circumscribed point; therefore, several points are positive in the same patient at the same time.

The tenderness occasionally is marked in two places: one at the base and the other at the tip of the appendix. This will give us a fairly good idea of the direction of the appendix.

If tenderness spreads from the right lower quadrant into other parts of the abdomen, it is a sign of the spreading of inflammation; if it spreads into all four quadrants, it is a sign of diffuse peritonitis; if it spreads still farther into both flanks and into both loins and can be elicited by rectal or vaginal examination, it is a sign of general peritonitis.

Deep tenderness may be elicited by digital compression of the McBurney, Morris, Lanz and Clado points or by palpation of the descending colon, which will reveal pain in one of the above mentioned points (Rovsing sign), or by inflation of the descending colon by air (Bastedo sign). It is, however, not advisable to employ the Bastedo sign for diagnostic purposes in acute appendicitis because there is a possibility that the inflated air may rupture the appendix. **Deep tenderness** is a more valuable diagnostic sign than the superficial type.

Rebound tenderness, that is, pain elicited after the hand compressing the left lower quadrant is suddenly released, is quite a constant symptom in all those cases in which deep tenderness is present (Blumberg sign). Rebound tenderness is present in about 72 per cent.

Rigidity or an increased tonus of the abdominal muscles is quite a constant symptom of peritoneal involvement (77 per cent). It appears after the pain and tenderness are already present. At first it is generalized, when pain and tenderness are "all over," but later it also "settles" in the right lower quadrant. The more pronounced the pathology and the more the parietal peritoneum is involved, the greater is the degree of rigidity. For this reason, the greatest rigidity is manifested in that part of the abdomen where the appendix lies. If it lies close to the anterior abdominal wall, rigidity of the right musculus rectus abdominis and of the right lateral muscles is the greatest. When there is a retrocecal appendix, the greatest rigidity may be in the lumbar muscles. In the case of a deep pelvic appendix, or if the appendix is well surrounded by omentum, there may be no rigidity whatsoever. However, in some cases this symptom is not reliable, because some patients cannot relax their abdominal muscles even if no pathology in the abdomen is present. That means that absence of rigidity is of greater diagnostic value than its presence.

It should also be remembered that rigidity of the abdominal muscles may be present in some pathological conditions located outside the abdominal cavity, such as pneumonia, pleurisy or coronary thrombosis. However, in such cases there will be no tenderness in the right lower quadrant.

FEVER. The rise of temperature is usually moderate; it starts between

three and twenty-four hours after the onset of the disease. The temperature at the beginning is about 99.5° F.; it increases every few hours and reaches its height at 102° F. in about twenty-four hours, at which point it remains for twenty-four to forty-eight hours and then gradually subsides, reaching the normal level by the end of a week. A sudden drop is a bad omen of approaching gangrene or perforation of the appendix. Cases with normal temperature are rare. The temperature usually starts without a chill. If it starts with a chill, it is probably not a case of appendicitis but if the clinical picture is suggestive of appendicitis, we are then dealing with a gangrenous appendix. However, this does not mean that every gangrenous appendix starts with a chill. As a matter of fact only few gangrenous appendices start with chills. If later in the course of the disease chills appear, it is a sign of some complications, such as pulmonary, peritoneal or vascular. If in the later course of the disease the temperature rises high and becomes irregular, it may be due to complications, such as pylephlebitis, subdiaphragmatic abscess, etc. Cases with initial high temperature (above 103° F.) with or without chills in the great majority of cases are not due to appendicitis.

PULSE. The pulse usually runs according to the temperature, that is 72 to 80 beats per minute with normal temperature and an increase of 10 beats per minute for each additional degree of F. This relationship is preserved during the entire length of the disease. Changes in this relationship have a definite significance. A slow pulse and a subnormal temperature (if all other acute symptoms persist) are suggestive of a gangrenous appendix. Very rapid pulse (130 to 140) is suggestive of peritonitis. The temperature in case of peritonitis at first is high, then becomes normal or even subnormal and later rises to 104° to 105° F.

However, it should be kept in mind that in nervous patients the pulse is liable to variations and it should be taken several times, particularly when the patient is off guard, otherwise one can give an erroneous interpretation of its frequency.

LEUKOCYTOSIS. The increase in the number of leukocytes may be slight and gradual or pronounced and sudden. It is important to register not only the total number of leukocytes per cubic millimeter but also the percentage of polymorphonuclear neutrophils. According to Sondern, an increase in the percentage of polymorphonuclear neutrophils is an index of the absorption of toxins, and the amount of leukocytes is the index of the patient's resistance to the absorbed toxins. In other words, the higher the leukocytosis, the better is the resistance of the patient, and the more the polymorphonuclears, the more severe is the infection. It is usually considered that a normal healthy individual has 6,700 leukocytes per cubic millimeter, with 62 per cent polymorphonuclear neutrophils. However, 10,000 leukocytes with 75 per cent of polymorphonuclear neutrophils is considered as a normal

amount by many. A slight increase of polynuclear neutrophils signifies mild infection; a great increase signifies severe infection. Slight leukocytosis and a slight increase of polymorphonuclear neutrophils signify mild infection with a good resistance. A great increase of leukocytes with a great increase in polymorphonuclears indicate severe infection with a good resistance. Slight leukocytosis and a great increase in polymorphonuclears signify a severe infection with a poor resistance. No leukocytosis and a marked increase in the polymorphonuclears signify severe infection and no resistance. A gradual increase in polymorphonuclear neutrophils with decrease of leukocytes signify increased infection with a decreased resistance. A gradual decrease in polymorphonuclear cells and a gradual decrease in leukocytes signifies improvement.

It should be borne in mind that the blood count alone is of no great diagnostic significance; but it becomes of the greatest diagnostic value when it fits into the clinical picture of acute appendicitis.

Of all the symptoms of acute appendicitis three are cardinal, namely, *pain, tenderness and rigidity*. They are the "triad" of the appendicular syndrome. But not all of these symptoms are always present. As a matter of fact, there is not a single symptom which is pathognomonic for appendicitis.

However, a study of all the symptoms, their appearance, course and interrelationship enables an experienced surgeon to arrive at a correct diagnosis even in an atypical case. In analyzing all the symptoms, one should always keep in mind that in the first forty-eight hours after the onset the symptoms do not run parallel to the pathological conditions present. At this time the anatomic-pathological symptoms are graver than the clinical manifestations; after forty-eight hours they run parallel and only then do the symptoms become a good guide as to the actual gravity of the pathology present.

SEDIMENTATION TEST. This test consists of letting citrated blood of the patient to stand in a column; then the erythrocytes settle down. If during an hour the erythrocytes settle with a velocity of 4 to 8 mm., it is considered normal; if it settles with a velocity up to 20 mm., it is considered moderately accelerated, and if above 20 mm., it is considered accelerated. On the first day of acute appendicitis the sedimentation test is normal in about 97 per cent in the male and in 89 per cent in the female. On the second day it is normal only in 85 per cent in the male and 68 per cent in the female. On the third day it is normal in 77 per cent in the male and 55 per cent in the female, and after the third day it is normal in 50 per cent in the male and in 44 per cent in the female.

The sedimentation test does not show an increase or diminution depending on the form of appendicitis (catarrhal, phlegmonous, destructive) and for this reason cannot be utilized for the diagnosis of the type

of appendicitis. There is no interrelationship between the degree of leukocytosis, number of eosinophiles and degree of sedimentation.

In the case of acute inflammation of the adnexa the degree of sedimentation is markedly increased and appears earlier than in the case of acute appendicitis. There is a different interpretation of this fact and it is used as a differential diagnostic point: If sedimentation is increased in the first day, it is probably not appendicitis but adnexitis; if, however, the sedimentation reaction becomes increased on the third day, it is a case of appendicitis.

X-ray has a very limited application for diagnosis of acute appendicitis. It goes without saying that no contrast medium should be given whenever acute appendicitis is suspected. However, flat x-ray plates are advisable for differential diagnosis in some conditions, which may resemble acute appendicitis, such as stones in the kidney or the ureter, biliary stones, some diseases of the lumbar vertebrae, such as spondylitis deformans, tuberculosis of the lumbar vertebrae, right-sided pneumonia, subdiaphragmatic abscess and, occasionally, pericarditis and mediastinitis. In the case of intestinal obstruction simulating appendicitis, x-ray is of great diagnostic value because it shows the ladder-like appearance of the small bowels.

Diagnosis

In the great majority of cases when most of the symptoms are present the diagnosis of acute appendicitis is not difficult. However, in the absence of some of the "cardinal symptoms" and, especially, when some symptoms are added, which arose due to complications, the diagnosis may become very difficult. In clinics headed by highly competent surgeons the percentage of mistaken diagnoses ranges from 8 to 22 per cent. It is a well known fact that cases of non-appendicitis are much more frequently mistaken for acute appendicitis than the vice versa and the ratio of this mistake is 10 to 1.

The diagnosis is based on the presence of a definite number of symptoms as described under "symptomatology" and on the presence of definite physical findings. The diagnosis does not depend on the presence or absence of some of the symptoms; the mutual relationship of symptoms has to be taken into consideration. There are many other lesions within or even outside the abdomen which may give identical symptoms. However, in different lesions the symptoms appear in a different order and different symptoms become the most prominent.

In order to differentiate acute appendicitis from all other conditions, one would have to review the differential diagnosis of nearly all possible pathological conditions within and even outside the abdomen. For this reason we will give only a few of the most frequently occurring intra- or extra-abdominal conditions:

1. **ACUTE CHOLECYSTITIS, CHOLELITHIASIS AND RUPTURED GALLBLADDER.** In the beginning of the disease they may simulate appendicitis because they also begin with pain in the epigastrium; however, they are more severe and are not colicky but boring and radiate to the right shoulder and to the right side of the back. Nausea, vomiting, fever and leukocytosis are present; however, tenderness and rigidity are pronounced in the right upper quadrant with its maximum intensity in the ninth right costal cartilage. **Biliary colic** is usually more severe than pain in appendicitis. **Rupture** of the gallbladder is accompanied by shock and peritonitis. The distinctive feature of pain in gallbladder disease is its more intense character, its radiation to the back, its tendency to remain around the starting place or to progress upward and not downward. Generally, the two conditions are not difficult to differentiate, particularly if one remembers that gallbladder diseases usually affect women past forty who are stout and that there is a definite past history of an idiosyncrasy to certain food.

2. **PERFORATED GASTRIC OR DUODENAL ULCER.** In typical cases the diagnosis is not difficult. In the case of perforated gastric or duodenal ulcer the pain is sudden and excruciating, there is a board-like rigidity of the abdomen, particularly in the upper portion, the pulse is rapid, temperature normal or subnormal at first, with a later rise due to peritonitis. In this stage the diagnosis is easy because appendicitis does not give such a severe picture. Slow leakage from a gastric or duodenal ulcer may produce symptoms resembling appendicitis. But even then the diagnosis, although more difficult, is still possible because the pain and rigidity are confined more to the upper abdomen and there is usually a history of an ulcer. Flat x-ray plates show air bubbles in the case of perforated ulcer.

3. **ACUTE SALPINGITIS** has a definite history of previous attacks of puerperal or venereal infection, of menstrual disorder or of frequent curettages. The pain is gradual at the onset and is confined to both sides of the lower abdomen. Vomiting is rare; the temperature is high, rising to 103° F., tenderness is mostly over the midsuprapubic region; muscular rigidity is not marked. However, in many cases the pain may be in the right side only and rigidity and tenderness also on the right side; then the diagnosis may be difficult. However, in the case of salpingitis tenderness on pressure in the cul-de-sac is present, particularly by pushing the cervix uteri with a finger upward. If pain is present at such a manipulation, it is a case of salpingitis. Temperature, pulse and leukocytosis are higher in salpingitis than in appendicitis. In the case of salpingitis usually both sides are tender and vaginal discharge and Gram-negative diplococci are ordinarily present.

4. **RUPTURED GRAAFIAN FOLLICLE OR RUPTURE OF THE CORPUS LUTEUM.** The rupture of a Graafian follicle or of the corpus luteum may

be accompanied by mild or by severe hemorrhage. In the case of mild hemorrhage the condition may be confused with acute appendicitis and in the case of severe hemorrhage with ruptured tubal pregnancy. In the presence of a rupture of the Graafian follicle or of the corpus luteum there may be nausea and vomitus; fever and leukocytosis are very often present and there will be tenderness, rigidity and rebound tenderness. The point of differentiation is that in the case of a ruptured Graafian follicle the pain starts in the right lower quadrant and then becomes generalized (in appendicitis the reverse is the case); it starts suddenly and vaginal examination is painful. The age of these patients is between fourteen and thirty-five years and most of them are multiparas.

5. **ACUTE PANCREATITIS.** The onset is sudden and is characterized by agonizing constant pain in the epigastrium and with symptoms of collapse. The blood pressure is low and the pulse is rapid. Extreme tenderness over the epigastric region is quite characteristic. When the retroperitoneal tissues are involved, the pain extends into the costovertebral angles. All these symptoms enable one to differentiate pancreatitis from acute appendicitis. In the differentiation between acute pancreatitis and any other intra-abdominal lesion the elevation of amylase in the patient's blood serum may serve as a valuable laboratory aid. The normal amount of amylase ranges from fifteen to thirty units, whereas in the case of acute pancreatitis it becomes elevated.

6. **RENAL COLIC** is characterized by colicky pain due to a stone in the kidney or in the ureter. It begins in the right lumbar region and radiates to the testicle and the penis or to the labia majora. There is no fever or leukocytosis. There is frequency of micturition and hematuria. An x-ray may show the stone. However, occasionally an inflamed appendix adherent to the ureter may give symptoms of renal colic and make the diagnosis very difficult. A flat x-ray plate of the kidney, ureters and bladder may show a shadow in the renal pelvis or along the course of the ureter.

7. **PYELITIS.** This is characterized by pain, fever and leukocytosis. The kidney is often sensitive. There is frequency of urination, chills and temperature up to 103° F. with a septic run. Pus in the urine is abundant. There is no rigidity or tenderness of the anterior abdominal wall. Vomitus, as a rule, is absent. However, in cases of acute appendicitis when the appendix is adherent to the ureter, pyuria may be present and pus disappears as soon as the appendix is removed. Pyelitis may be ruled out by urine examination, pyelography and palpation.

8. **DIETL'S CRISES**, which are attributed to a twisting of the movable kidney on its ureteral pedicle with compression of the blood vessels and nerves, produce attacks of pain in the right lumbar and abdominal regions. The pain is colicky, often accompanied by nausea and vomiting;

fever rises to 101° F. and frequently there is tenderness and rigidity of the right lower abdomen. The points of differentiation are: This condition occurs mostly in women, there is no leukocytosis and no point of tenderness; symptoms come and disappear quickly; the pain is relieved if the patient lies down and especially if the hips are elevated.

9. **TUBAL PREGNANCY IMMEDIATELY BEFORE RUPTURE**, at the time of rupture and after the rupture gives many symptoms similar to appendicitis, such as pain, tenderness, rigidity and leukocytosis. For differentiation before the rupture the most important is the history of menstrual irregularities; at the time of rupture, shock, bloody discharge from the vagina, thready pulse, pallor and lowered blood pressure; after rupture a pulpy mass in the cul-de-sac may be felt.

10. **TORSION OF THE PEDICLE OF A RIGHT OVARIAN CYST**. Confusion is easy because this condition elicits severe abdominal pain, nausea, vomiting, rapid pulse, fever and tenderness. The diagnosis will be cleared up by a bimanual examination which may reveal a tumor, by a history of the presence of an ovarian tumor and by the low position of the affected area. The tumor is usually felt, because small size cysts ordinarily do not produce torsion of the pedicle.

If, however, the torsion lasts for several hours, the blood supply is shut off, gangrene ensues and pain disappears. At this stage diagnosis of appendicular abscess can be made easily.

11. **DIVERTICULITIS**. It may be necessary to differentiate from Meckel's diverticulum (congenital type) or from the inflamed diverticula of the large bowel (acquired type). In the case of acute diverticulitis of the large bowel, it is usually at the sigmoid and symptoms are confined to the left side; but when they become more generalized, the symptoms may be confused with appendicitis. The fact that diverticulitis occurs in middle age and that pain is on the left side may serve as diagnostic points for differentiation. One has to keep in mind that a long appendix can reach the left side and give all the symptoms of diverticulitis. Meckel's diverticulitis produces the same clinical symptoms as acute appendicitis and at the beginning of the disease it is extremely difficult to differentiate between these two conditions.

12. **THROMBOSIS AND EMBOLISM OF THE MESENTERIC VESSELS**. This condition is more often confused with intestinal obstruction than with appendicitis. It is characterized by a sudden, excruciating pain in the umbilical region, followed by vomiting and collapse. There is no passage of stool or gas; if, occasionally, the bowels move, there is a bloody, watery or mucoid stool. The pulse is usually weak and rapid and the temperature subnormal. The differentiation usually is not difficult, yet in cases which start not so acutely it may become very difficult. The points of differentiation may be: (1) History of a previous embolism in some other part; (2) colicky pain in the abdomen; (3) marked fall of

Treatment

The treatment of acute appendicitis essentially is surgical. However, there are instances in which medical treatment is preferable. As long as the appendix lies free and is not surrounded by intra-abdominal structures which would shut it off from the free peritoneal cavity and as long as the inflammatory process is progressing—the appendix should be removed. But when protective adhesions are formed, the time for operation has passed and conservative treatment becomes less hazardous. In average cases these protective adhesions (mass-formation) are formed within forty-eight to sixty hours after the onset. For this reason surgery is indicated within the first forty-eight to sixty hours after the onset and conservative treatment if more time has elapsed. However, it should be remembered that these hours represent the time only fairly accurately when mass-formation takes place but the real decision to operate or not depends on whether or not an abdominal mass is present. For this reason, as soon as the diagnosis has been made one should ascertain whether an abdominal mass is present. If it is, conservative treatment should be instituted.

However, if the mass is not felt the patient should be examined again under general anesthesia. In many cases the mass, which was not felt while the patient was awake becomes palpable after the patient is completely relaxed under deep general anesthesia. If a mass is palpable under deep anesthesia, no operation should be done. If no mass is felt under deep anesthesia and a mass is found, after the abdomen has been opened, the appendix is removed only if it lies superficially, is easily accessible and does not necessitate the breaking of adhesions. If, however, the appendix is included in a mass and it can be reached only by breaking the surrounding adhesions, the appendix should not be removed and a cigarette drain should be placed in the free peritoneal cavity and the abdomen closed.

The management differs depending on the various phases of pathology found, namely, if the condition is confined to the appendix only, if there are manifestations of spreading peritonitis, general peritonitis, mass-formation or abscess-formation. In addition, there might arise numerous complications some of which may need surgical management.

If the pathology is confined to the appendix only, or if there are only mild symptoms of spreading peritonitis which do not involve a large area, simple appendectomy should be performed.

Management of Acute Appendicitis in which Pathology is Confined to the Appendix Only

After it is decided that the patient should be operated upon many points have to be taken into consideration:

1. Choice of the abdominal incision;
2. Location of the cecum;
3. Location of the appendix;
4. Technic of removal of the appendix;
5. Question of drainage.

1. CHOICE OF THE INCISION (Fig. 166). There is not a single incision

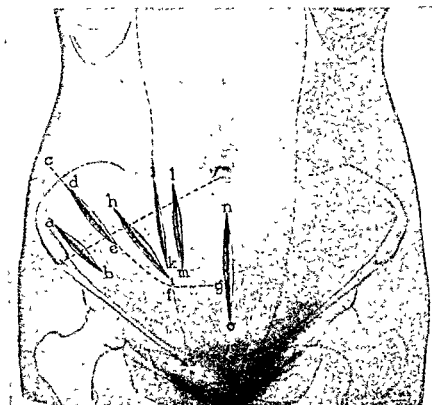


FIG. 166. Incisions employed for appendectomy. *ab*. Sonnenburg incision. *de*. McBurney incision. *defg*. Harrington incision. *jk* and *lm*. Battle incision (Jalaguier, Lennander, Kammerer, Senn incisions). *no*. Midline incision

which serves all purposes. For acute appendicitis one of two incisions are usually employed—either McBurney's or any of the right pararectus incisions which are known under different names (Jalaguier, Battle, Kammerer, Lennander, Senn). The majority of surgeons consider that the McBurney incision should be employed for all acute cases in which there is no intention on the part of the surgeon to explore the abdominal cavity and all that the surgeon wants is to "get in quick and get out still quicker." In instances in which the surgeon expects to have some technical difficulties (very obese patients), or he believes that the abdominal cavity should be explored, a right pararectus incision should be used because it offers a better operative field. The contention of some surgeons that a right pararectus incision should not be used in acute cases,

because it gives a higher percentage of mortality on account of the easier spreading of peritonitis, is without justification. There is ample statistical evidence that peritonitis does not occur more frequently when a right pararectus incision is used.

We are in full accord with this latter view. If we consider that the base of the appendix lies at the point of the cecum, which holotopically corresponds to the point where the lateral border of the right musculus rectus crosses the spino-umbilical line, we do not see any reason why an incision passing through this point should promote the spreading of peritonitis.

Technic of McBurney Incision (Fig. 167). A point is selected on the spino-umbilical line at a distance of one and a half inches from the superior anterior iliac spine. A skin incision three inches long is made through this point perpendicularly to this line. Two inches of this incision are above the spino-umbilical line and one inch below. After the skin incision, the superficial and deep fascia are incised, the external oblique muscle is exposed above the spino-umbilical line and aponeurosis of the same muscle below this line. The external oblique muscle and its aponeurosis are split in the same direction as the skin incision.

Small retractors separate the lips of the split muscle and expose the internal oblique muscle, which is split in the direction of its fibers. The hooks that retracted the lips of the external oblique muscle are removed and then reinserted to separate the lips of the internal oblique muscle and to expose the transversus abdominis muscle. The fibers of this muscle are separated by a very slight incision of the fascia covering the transversus abdominis muscle. Then the jaws of a curved artery forceps are inserted until the transversalis fascia is reached when the jaws are spread open. After this is done, the transversalis fascia immediately protrudes; it is caught between two tissue forceps and cut together with the peritoneum in a direction perpendicular to the midline.

This incision is excellent in a great number of cases; the appendix can be easily removed and there is very little damage to the anterior abdominal wall. However, in some cases, due to the smallness of the incision, it is difficult to locate not only the appendix but even the cecum; in some instances the cecum can be located but the appendix is difficult to deliver; then it is necessary to enlarge the incision. It is usually enlarged in one of two directions depending on the location of the appendix. If it lies high up under the liver, the incision is prolonged at the upper end in the direction of the line of the incision already made. After the skin, superficial and deep fascia are cut in this direction, the fibers of the external oblique muscle are split in the same direction. The lips of this muscle are retracted and the internal oblique, the transversus abdominis muscles and the transversalis fascia with peritoneum are cut in a direction perpendicular to the direction of the fibers of the external

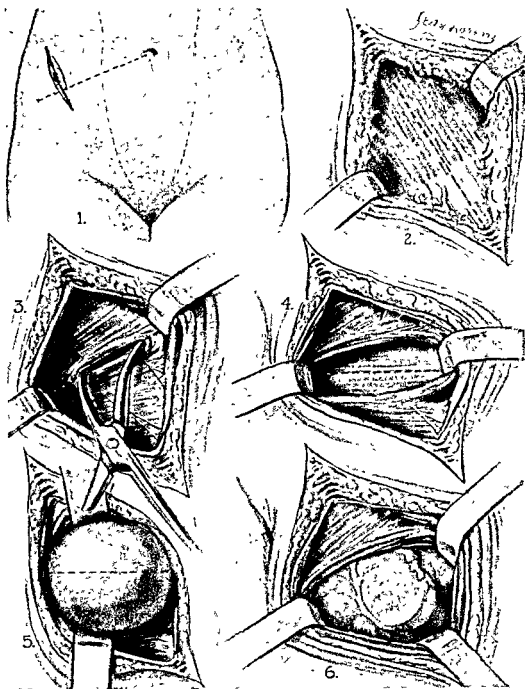


FIG. 167. Technic of McBurney incision. 1. Skin incision. 2. Splitting of fibres of external oblique muscle and its aponeurosis. 3. Splitting of fibres of internal oblique muscle. 4. Splitting of fibres of transversus abdominis muscle. 5. Cutting the transversalis fascia and peritoneum. 6. The cecum is seen in the abdominal cavity.

oblique muscle, starting from the upper angle for a distance of an inch or an inch and a half. This will result in a good exposure of the field.

If the appendix lies in the pelvis and it is necessary to enlarge the incision downwards, the Harrington incision (Fig. 168) will afford a good

exposure. This incision is made in the following manner: From the lower end of the previous McBurney incision the new incision is carried downward and inward until it reaches the lateral border of the right

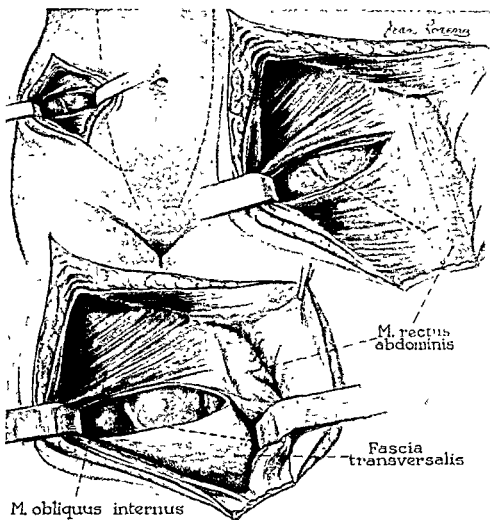


FIG 168. Harrington incision. Upper left. The skin incision is prolonged downward and medialward. Upper right. Cutting of the internal oblique and the most lateral part of the anterior sheath of the m. rectus. Lower picture. The m. rectus is retracted medialward. Cutting through the transversalis fascia.

rectus muscle; the anterior sheath of this muscle is cut transversely for one inch, just sufficient to expose the lateral border of the rectus muscle and to enable the introduction of a retractor to draw this muscle inward. Then the posterior sheath with the transversalis fascia and peritoneum (or the transversalis fascia and the peritoneum if the posterior sheath did not reach so low) are incised transversely to the midline. This will afford a good exposure.

As soon as the abdomen is opened, one has to proceed with the location of the cecum, which is the preliminary step in locating the appendix. In some cases the appendix appears in the wound so that there is no necessity for locating the cecum. However, in the great majority of cases one has to search for the cecum in order to locate the appendix. Usually it is an easy procedure and one finds the cecum by inserting the index finger and the thumb into the right iliac fossa. However, difficulties are encountered occasionally because the cecum may be covered by loops of small bowel and all the time such a loop appears in the abdominal incision. When this occurs there are few methods which facilitate the location of the cecum.

MURPHY METHOD OF LOCATION OF THE CECUM. The index finger of the right hand is inserted through the abdominal incision and carried behind the anterior abdominal wall to the right until the lateral wall is reached; the finger then is carried along the lateral abdominal wall until the posterior abdominal wall is reached and then medially until it reaches the pouch formed by the junction of the parietal peritoneum of the posterior abdominal wall with the cecum. The latter is then grasped between the thumb and the index finger of the right hand.

This is a good method and is efficient for the majority of cases. However, there are occasions when this method is not satisfactory, namely, when the cecum lies close to the posterolateral side of the abdomen. Under such circumstances there is no pouch formed between the abdominal wall and the cecum, and the exploring finger, after leaving the lateral abdominal wall, will traverse across the posterior wall actually in front of the cecum and may pick up any loop of bowel but the cecum. If this occurs there is another method known as the Monks' method, which will enable one to find the cecum by locating first the terminal portion of the ileum at the distance of one to four inches from the ileocecal junction.

MONKS' METHOD (Fig. 169). The index finger of the right hand is placed on the right side of the brim of the pelvis. This finger is then carried upward along the pelvic brim and still farther upward in the same direction until a pouch is encountered. This pouch is formed by the attachment of the lowermost portion of the mesoileum to the posterior abdominal wall. Then in front of the finger the terminal four inches of the ileum will lie; the latter is drawn outside between the thumb and the index finger; as soon as the terminal ileum is located, the cecum is found one to four inches away from it.

In acute appendicitis we prefer to locate the cecum by merely picking it up between two fingers. If we are unable to locate it in this manner, we employ the Murphy method; if this fails, we use Monks' method. The reason for our preference for this succession of methods is that in each previously mentioned procedure we come less in contact with the ab-

dominal viscera and therefore there is less danger of contamination.

There are, however, instances, when using Monks' method one will be unable to find the terminal portion of the ileum. This will be in those rare cases in which the cecum failed to descend from the subhepatic

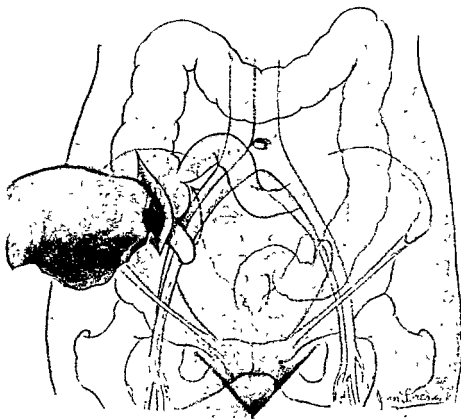


FIG. 169. Location of terminal ileum by Monks' method.

region into the right iliac fossa. However, this does not hold against Monks' method. On the contrary, it gives additional value to this procedure because it not only enables one to locate the terminal ileum and cecum, when they are present in their usual location, but also shows when the terminal ileum and cecum are absent in this location.

One should also bear in mind that extensive adherence of the terminal ileum to the right iliac fossa by a Lane band may occasionally produce difficulties in locating the terminal ileum by Monks' method.

Inability to locate the terminal ileum and cecum by Monks' method is a sign that the incision should be enlarged in an upward and lateral direction.

LOCATION OF THE APPENDIX. The base of the appendix is situated on the posterior wall of the dome of the cecum in 90 per cent of all cases. In order to locate it, the dome of the cecum is turned upward, so that its posterior wall faces anteriorly and the appendix becomes visible. If the appendix is not seen, one has to look for the ileocecal fold ("Bloodless fold of Treves") (Fig. 165). This fold runs from the antimesenteric border of the terminal ileum, plasters against the cecal wall and fuses with the mesoappendix. Naturally, as soon as the mesoappendix is located, the appendix is easily found.

There is another method of locating the appendix, namely, by tracing the three taenia longitudinalis coli until they cross each other, because the appendix springs from the point of their crossing.

Usually, it is easy to locate the appendix by one of these methods. However, occasional difficulties may arise. They may be due to one of three causes:

- A. Absence of the appendix—acquired or congenital.
- B. Hypoplasia of the appendix.
- C. Hidden position of the appendix which otherwise is of a normal size.

A. Absence of the Appendix. This may be either acquired or congenital. Acquired absence of the appendix is a well known fact. Occasionally, while operating upon a patient for acute appendicitis, one discovers that the appendix sloughed off the cecum and lies either free in the peritoneal cavity or surrounded by omentum or loops of bowel. Cases have been reported of patients having acute attacks of appendicitis who refused operation. The patients had palpable masses in the right lower quadrant and a stormy recovery. Years later some of these patients were operated upon for some other abdominal condition and no appendix was found. It is reasonable to presume that the appendix sloughed off in a previous attack but the patient survived the attack. However, it should be remembered that an absence of the appendix of the acquired variety is rare and congenital absence of the appendix is still rarer.

Up to date there have been only 50 odd cases of the congenital variety reported. In 1925, Dorland in an excellent article on the subject reported thirty-six cases to which he added his own case. In 1930, I collected seven cases that appeared after Dorland's publication and added my own two cases, which were the forty-fifth and forty-sixth cases. Green and Ross published a case in 1933, Sindoni in 1933, Louyot, Richon and Lacourt in 1934, Darrah in 1935, Caminiti in 1937, Balice in 1938, Rambo and Lasky in 1938, thus bringing the number of cases to fifty-three.

In some the appendix was not found on the operating table and for this reason one cannot be certain whether the appendix was really ab-

sent. Another group of absent appendices were those which were not found on the dissecting table. They are much more reliable than the cases of the first group but still are not absolutely reliable. Several times while dissecting cadavers we did not find the appendix. The most careful palpation could not reveal it and it was only after the serosa of the

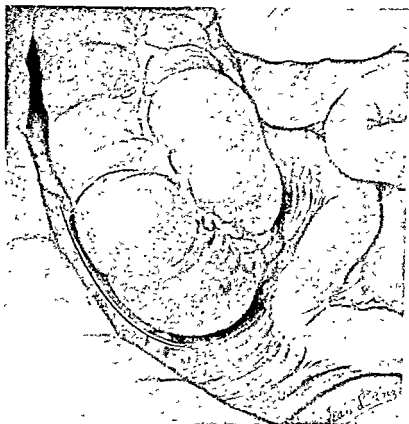


FIG. 170. Location of the appendix if Lane's band is present.
Mobilization of the cecum (Spivaek's rule).

cecal wall was incised, starting at the point where the base of the appendix has its usual position, that an appendix, perfectly normal in size and appearance, was found (Fig. 172, insert). The reason why the appendix was not palpated was that since it was not inflamed it had the same consistency as the cecal wall and since it was covered by serosa it was invisible, hence the reports of "congenital absence of the appendix" in cases not found on the operating table.

But even if we consider that all cases reported as an absence of the appendix, acquired or congenital, actually were cases of absence (and we do not concede that this is the case), they are so rare that for all practical purposes we may consider that they hardly ever occur and for this

reason if one cannot find the appendix, one should dismiss the thought of its absence and think of one of the remaining two possibilities:

B. *Hypoplasia of the Appendix*. The average length of the appendix is about three inches. However, in its extremes the length may vary con-

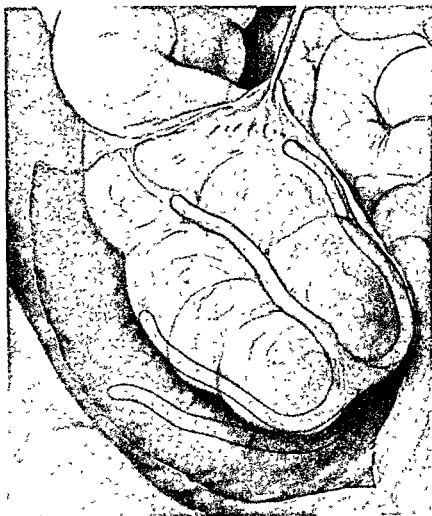


FIG. 171 Different positions of the retrocecal appendix.

siderably. Cases are known of the appendix being $12\frac{1}{2}$ inches (33 cm.) long and of 2 mm. ($1/13$ of an inch) short. However, appendices shorter than one-half an inch are infrequent. When they are small, they may have an appearance of one of the appendices epiploicae. However, the differentiation is easy. If the structure in question originates from the point of crossing of the three taeniae longitudinales, it is the appendix. Histologic examination, of course, will corroborate or disprove this.

C. *Hidden Position of the Appendix*. In the great majority of cases the base and the proximal portions of the appendix lie retroceally and

solution is then placed medialward and downward and another one upward and lateralward from the cecum. This "walls off" the cecum with the appendix from the rest of the peritoneal cavity. This "walling off" is advisable only when the abdominal incision is large enough to permit the

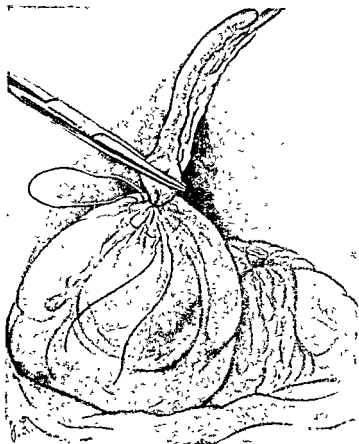


FIG. 174 Technic of appendectomy. Insertion of a purse-string suture on cecum around the base of the appendix.

small bowel to protrude outside the abdominal cavity and come into contact with the cecum and appendix or if the cecum and appendix are situated deeply and cannot be delivered outside the abdominal cavity. If the appendix and the adjacent portion of the cecum can be delivered into the small size abdominal incision so that they close the opening snugly, there is no need for placing laparotomy sponges into the peritoneal cavity. All that is necessary then is to wrap the protruding portion of the cecum with moist laparotomy sponges so as to prevent any soiling of the peritoneal cavity and of the thickness of the abdominal wall due to manipulations on the cecum and the appendix. Next, the appendiceal artery, which is a branch of the ileocolic artery and runs along the free border of the mesoappendix, is ligated either by clamping

the mesoappendix, cutting it and then tying it, or by carrying a thread around the base of the mesoappendix on an aneurysm needle or on an artery forceps (Fig. 173, insert).

The next step is the actual removal of the appendix and the management of the appendiceal stump.

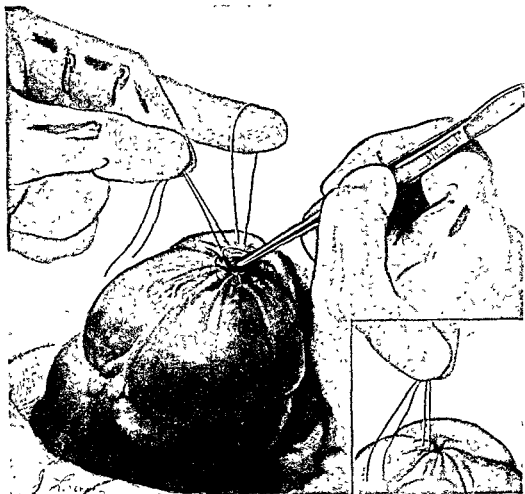


FIG. 175 Burying the appendiceal stump in the cecal wall.

There are several methods suggested all of which have their strong and weak points. We believe that there is not a single method which fits for all occasions. There are conditions when one method should be preferred to another, whereas in a second group the reverse is the case. For this reason we give the description of several methods:

a. REMOVAL OF THE APPENDIX AFTER PRELIMINARY LIGATION OF ITS BASE AND DROPPING THE LIGATED STUMP INTO THE FREE PERITONEAL CAVITY (Fig. 176 1). This method is the earliest type and the first appendectomy performed by Krönlein in 1884 was with this procedure. It was used by all surgeons until 1888 when Frederic Treves

suggested the peritonization of the stump of the appendix by a peritoneal cuff. The latter method superseded the former. Senn, in 1889, was the first American surgeon to advocate inversion of the appendiceal



FIG. 176. Various methods of managing the appendicular stump. I Tying the stump and leaving it in the peritoneal cavity. II. a Burying the stump in the cecal wall. b. Cross section showing the appendicular stump lying in the cecal wall. III. a. Dawbarn method of pushing the untied stump into the lumen of cecum. b. The same in cross section.

stump. In 1904, M. G. Seelig again revived interest in the "simple ligation method." He contended that in the "ligation and burying"

method there was the danger of abscess formation on the wall of the cecum which is transformed into a closed chamber in which the stump lies. Such an abscess could rupture into the free peritoneal cavity. This view is shared by quite a number of highly competent surgeons. M. G. Kamenchick, basing his report on the material secured at the Leningrad Institute of Urgent Surgery, gives a statistical review of 1,944 patients operated upon for acute appendicitis in the years 1932 and 1933. Of this number 1,127 persons were operated upon by mere "ligation," 559 by the "ligation and burying" method and 258 by the method of "inversion without ligation of the stump." In none of these methods were there complications that were absent in other methods, with exception of the "inversion" method in which hemorrhage occurred in one case.

b. REMOVAL OF THE APPENDIX AFTER PRELIMINARY LIGATION OF ITS BASE AND BURYING THE STUMP IN THE WALL OF THE CECUM (Fig. 176ii)

This method was introduced in the United States by Nicholas Senn in 1889 and became the most popular. The advocates of other methods offer some objections to the use of this procedure. The main objection to this method is that an infected stump (even if it was sterilized by carbolic acid or cautery) is buried in a closed cavity and that abscess formation is possible. This abscess occasionally breaks into the free peritoneal cavity producing peritonitis, or through the skin, producing sinuses, or simultaneously into the lumen of the cecum and through the skin outside producing a fecal fistula (Fig. 177). Occasionally the omentum becomes adherent at the point where the stump is buried. The reason for this is that in about 80 per cent of call cases in which a purse-string suture is made it becomes infected and thus produces adhesions. However, it should be stated that these complications are very infrequent and the followers of this method claim that it is surgically more correct than the first method described. They claim further that it would be dangerous to leave an infected stump in the free peritoneal cavity, that the ligature may slip off and produce fatal peritonitis and that a loop of bowel may adhere to the raw surface of the stump of the appendix and cause intestinal obstruction.

c. REMOVAL OF THE APPENDIX WITHOUT LIGATION OF ITS BASE AND INVERSION OF THIS NONLIGATED STUMP INTO THE LUMEN OF THE CECUM AND CLOSURE OF THE CECAL OPENING BY A PURSE-STRING SUTURE (Fig. 176 iii). This method was introduced in 1891 by Dawbarn. It consists in the placing of a crushing clamp at the base of the appendix and another one distally from it, making a seromuscular purse-string on the cecum as in the second method, "shaving off" the appendix between the two clamps, inverting the crushed stump into the lumen of the cecum after withdrawing the proximal clamp and tying the ends of the purse-string suture, thus closing the hole in the cecum. It eliminates

some objectionable features of the two previous methods but creates some new objectionable features. To begin with, it retains one of the disadvantages of the previous method, namely, the introduction of the purse-string suture on the cecal wall. Although this suture is supposed

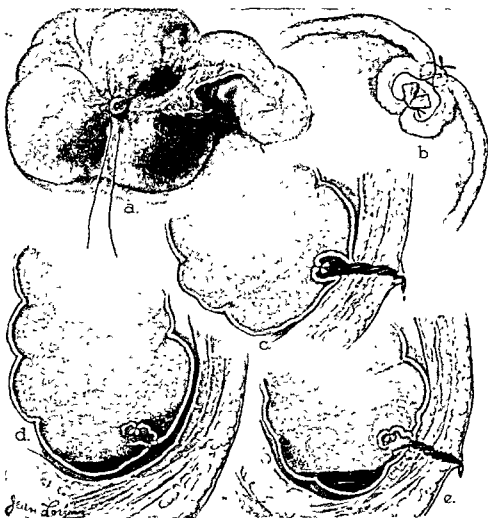


FIG. 177. Complications which may arise in management of appendicular stump. a. Burying the stump in the cecal wall. b. Cross section of the same. c. Formation of a sinus. d. Rupture of the abscess in the cecal cavity with spontaneous recovery. e. Rupture of the abscess in the cecal lumen and outside with formation of a fecal fistula.

to be seromuscular, nevertheless it has been shown that in about 90 per cent of all cases one or more bites penetrate the mucosa and the suture becomes contaminated. A still more important objection is that from time to time hemorrhage occurs from the end of the nonligated crushed appendicular stump. As discussed already, the blood supply to the appendix is derived from the appendicular artery and runs in the free bor-

der of the mesoappendix, sending short branches to the appendix (Fig. 165). However, in some cases the main appendicular artery is absent and the blood supply to the appendix is derived from the cecal artery and traverses to the appendix over its base usually along its mesenteric border. If the stump is ligated, the ligature checks the possibility of bleeding; if there is no ligature applied, there may be a possibility of a hemorrhage and, indeed, cases have been reported in which hemorrhage occurred.

However, we believe that this objection can be easily overcome by two manipulations: first, the base of the appendix should be crushed and with it the blood vessels will be crushed; second, by making a purse-string suture care should be taken so that the branch running to the appendix either is taken in a loop or the threaded needle is passed in front and not behind this branch. At any rate, this hemorrhage occurs seldom even if no special precautions are taken, and certainly should not occur if the above precautions are followed. For this reason we believe that the Dawbarn method is to be preferred to any other technic of dealing with the stump, whenever there is a free choice.

In conclusion we wish to repeat the statement with which we started the discussion on the management of the stump, namely, that there is not a single method of handling the stump that will suit all cases. Thus, in acute appendicitis with a pronounced edematous wall of the cecum in which a purse-string suture cannot be inserted, neither the b nor the c method can be employed. When there is a very thick edematous appendix in which the wall of the cecum is not much changed, the Dawbarn method will not be applicable, since this procedure presupposes crushing the stump and this is contraindicated in such a type of appendix. We may speak of preference in using one or another method in dealing with the stump, but there are cases when we are forced to use a method which we would otherwise consider as a second or third choice.

RETROGRADE APPENDECTOMY. By this term is meant a procedure in which the appendix is separated at its base from the cecum as the first step of the operation, and the remainder of the appendix is removed in subsequent steps. This type of operation is performed when the distal portion of the appendix is adherent to the surrounding structures by dense adhesions. When the whole appendix is imbedded in strong adhesions and its separation carries the danger of perforating the appendix, it is a good plan to remove the appendix by the retrograde method. The technic of this method is as follows:

Step 1. The abdomen is opened and the cecum is located by one of the methods described above.

Step 2. Two crushing forceps are placed at the base of the appendix perpendicularly to its long axis (Fig. 178) and the appendix is cut between the two forceps by cautery or knife. If it is cut by knife, the two

stumps are carbolized. The base of the appendix is then managed by one of the methods described previously for handling the stump (Fig. 176).

Step 3. The appendix is stretched by the artery forceps which holds its severed end and straight arterial clamp is applied to its mesentery. If the latter is too long, or if the entire appendix cannot be delivered at once, another artery forceps is applied to the remainder of the meso-

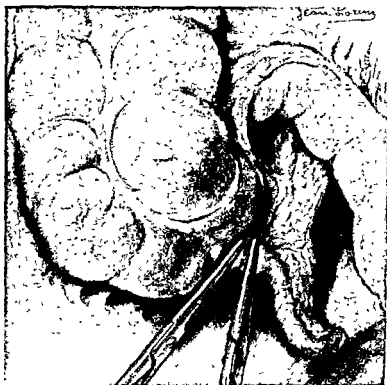


FIG. 178. Retrograde appendectomy. Two artery forceps clamp the base of the appendix.

appendix, which is then cut between the artery clamp and the body of the appendix (Fig. 179). The clamped mesenteric stump should be ligated.

SUBSEROUS APPENDECTOMY (Figs. 180, 181). This operation consists in enucleating and removing the mucous tube of the appendix and leaving the muscular and the serous coats. It is indicated in cases in which the appendix lies in dense adhesions and when the separation of the adhesions from the surrounding structures may injure them. This operation was first performed by Poncet in 1899, and was reported the same year by Vignard. Poncet coined the term "subserous appendectomy." Poncin, in 1901, and Delore, in 1902, reported several cases of this type of appendectomy. In 1905, Isaacs, of New York, reported several cases

of subserous appendectomy, which he called "decapsulation of the appendix." Recently some surgeons advocated the use of this method not only for cases in which dense adhesions are present but even when the appendix is free. However, we believe that this method should be employed only in those cases in which dense adhesions are present and

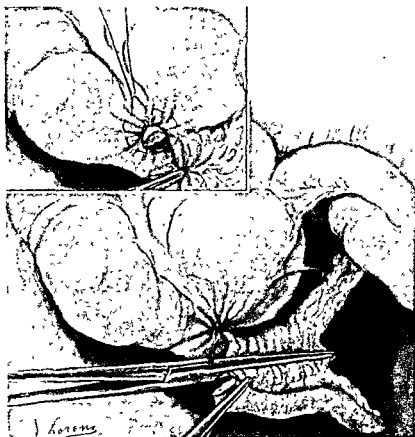


FIG. 179. Retrograde appendectomy. Main picture: A forceps clamps the mesoappendix. Insert: Burying the appendiceal stump into the cecal wall by a purse string suture.

not in cases in which the appendix can be readily separated from the surrounding structures.

Technic. Step 1. The cecum and appendix are walled off from the rest of the peritoneal cavity.

Step 2. The mesoappendix at the tip of the organ is grasped by an artery forceps and the appendix is put on the stretch. A linear incision is made, cutting through the adhesions, serosa and muscularis of the appendix until the mucosa is reached. This incision is made along the entire length of the antimesenteric border (Fig. 180). Both seromuscular lips are reflected so as to isolate the mucous tube in its entire circumference at least in one place. This should be done close to the tip, because this is the safest point in case the mucosa is accidentally injured.

A hook is placed around this completely isolated mucous tube, and the isolation is continued until the base of the appendix is reached (Fig. 181), which is then crushed and cut between two clamps. The proximal crushed end is tied and the stump sterilized, after which it is buried by a purse-string suture. The seromuscular lips are then sutured to each other (Fig. 181, insert).

Drainage. The question of drainage has been a controversial one for many years and even now the surgeons are not in accord on many



FIG. 180. Subserous appendectomy. Incision of the serosa over the appendix

points. In the early days of appendectomy there was a tendency to drain the abdominal cavity in all cases in which turbid fluid was found in the free peritoneal cavity, in cases of perforated or gangrenous appendices or in cases in which fibrous exudate was on or around the appendix. As a matter of fact, drainage was encouraged for all those cases in which the appendix showed something more than a mere catarrhal inflammation by the well known maxim of Lawson Tait: "when in doubt—drain." However, in the early part of the first decade of this century there appeared reaction against this indiscriminate drainage. Yates, of Milwaukee, as early as in 1904 showed that a drain introduced into the

general peritoneal cavity of a dog after six hours is walled off and does not communicate with the general peritoneal cavity. He showed that carmine, injected into the free peritoneal cavity elsewhere, did not pass through the tract produced by the drain. In the presence of infection this "walling off" was taking place in a shorter period of time than in a normal peritoneal cavity. He also showed that in cases in which a drain was inserted the peritoneum harbored micro-organisms for a longer



FIG 181. Subserous appendectomy. Peeling out the mucous tube of the appendix.

period of time than the peritoneum in nondrained cases. The work of other investigators showed that a drain, acting as a foreign body, lowers the immunity of the peritoneum against infection. It also causes definite pressure on the surrounding small bowels which diminishes the peristalsis or increases the stasis of the intestinal contents, thus increasing the toxicity of the patients. Some investigators showed that when the drain is inserted a profuse purulent discharge from such a wound is observed. This is not formed from within the peritoneal cavity but from the supuration of the abdominal wall. From there it collects along the drain-tract into the depth of the abdominal wound, thus aggravating, instead of clearing, the infection. Other investigators described the occurrence

of early and late postoperative herniae. In some cases pressure-necrosis of the intestinal wall produced a fecal fistula and in still other cases secondary intra-abdominal abscesses were formed.

All these facts are responsible for an increase in immediate postoperative mortality and in late morbidity. Indeed, there are statistical data comprising thousands of cases which show that mortality is considerably lower in those cases of equal gravity in which no drains were placed and the abdomen was closed blindly. Thus it is evident that in the case of a gangrenous or perforated appendix or when turbid exudate is present in the free peritoneal cavity the abdominal cavity should not be drained. Whenever the appendix is removed and the peritonization of the damaged area is accomplished no drain should be inserted. If however, there is an abscess formation, or the appendix cannot be removed, or peritonization of the affected viscus cannot be accomplished, the abdominal cavity should be drained. However, the muscular and fascial layers of the abdominal wall cannot combat infection as efficiently as the peritoneum does it. Therefore, whenever there is a suspicion that the abdominal wall has been contaminated, it is advisable after completely suturing the peritoneum to dust the thickness of the abdominal wall with sulfathiazole and then to close it. If the patient runs a temperature postoperatively we give him daily intravenously four grams of sulfathiazole or sulfadiazine or 300,000 units of penicillin.

Management of Spreading Peritonitis

By this term is meant a condition in which there is no tendency toward localization of the inflammatory process but only a portion of the peritoneal cavity in the vicinity of the appendix and cecum is inflamed. In such cases after the abdomen is opened one finds that the serosa covering the small bowel is still shiny, the walls are not edematous and the abdominal cavity may or may not contain seropurulent material. When there is a spreading type of peritonitis, we remove the appendix. All raw surfaces are peritonized and we close the abdomen without drainage of the peritoneal cavity. If the abdominal wall has not been contaminated, we close it in layers by a series of interrupted sutures. If we suspect that we have contaminated the abdominal wall, we dust it with sulfathiazole powder and close the wound. If the patient runs a temperature we administer daily intravenously four grams of sulfathiazole or sulfadiazine or 300,000 units of penicillin.

Management of Diffuse Peritonitis

If a picture of diffuse peritonitis is present, it is not advisable to operate. There is ample statistical proof that such patients do considerably better by means of conservative treatment, namely, by decompression of the stomach and small bowel by continuous suction (Wangensteen

method), intravenous administration of sulfa drugs and penicillin and hypodermic administration of morphine. This drug not only diminishes pain but also, taken in moderate doses, increases peristalsis, thus diminishing intestinal stasis. The latter increases toxicity which is one of the chief causes of death in peritonitis. The intravenous injection of 3,000 or 4,000 cc. of normal salt solution with 10 per cent glucose prevents dehydration. It is also advisable to decompress the colon by inserting a

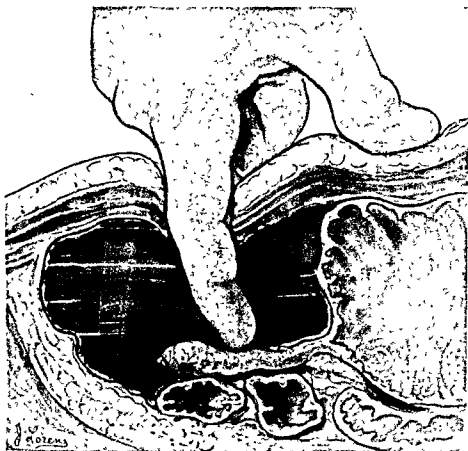


FIG. 182. Management of appendicular abscess. Extreme care should be taken not to break the wall of the abscess.

rectal tube. Some surgeons recommend appendicostomy or cecostomy. However, we prefer not to operate at all and to treat the condition conservatively; but if the abdomen has been opened because the extent of pathology had not been clear without laparotomy and diffuse peritonitis had been found we do a cecostomy according to the Witzel method. This treatment is discussed in more detail in the chapter on peritonitis.

Management of Appendicular Abscess (Figs. 182, 183, 184)

There is a great deal of controversy in regard to the management of appendiceal abscess. One reason for this is the confusion caused by the

name. Some apply the term "abscess" to a conglomerate of bowels which properly should be called "mass formation." Others call an abscess a well walled-off mass in which suppuration has taken place and which consists of pus and débris.

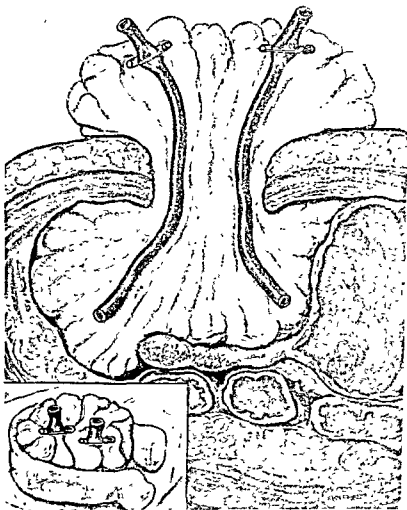


FIG. 183. Drainage of appendicular ab-cess (After Bosh-Arana).

We believe that any mass in which no pus is present should be called "infiltrate" or "a mass formation" and reserve the name "abscess" to a conglomerate which actually contains pus. The treatment differs depending on whether we deal with an "infiltrate" or with an "abscess." In the case of an infiltrate, we believe in treating the patient conservatively.

The patient should be placed in Fowler's position. For the first three days nothing should be given orally and normal salt solution with 10

per cent glucose should be given intravenously; then liquids may be added by mouth. Sulfa drugs and penicillin should be given intravenously from the very first day. Morphine should be administered and a rectal tube inserted in case of gas pain. Cold applications or ice bag should be applied to the abdomen. With this method of treatment the infiltrate in the majority of cases disappears within seven to fifteen days. However, in other instances it does not disappear but becomes larger and

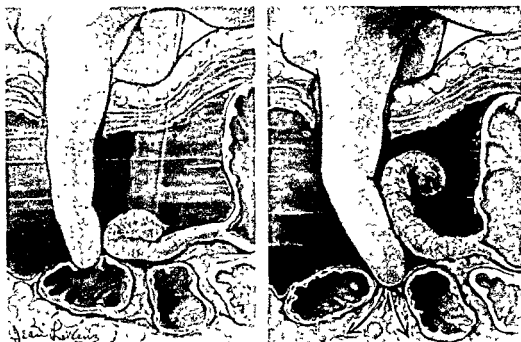


FIG. 184. Management of appendicular abscess. Left. Possibility of breaking the intestinal wall of the abscess with formation of a fecal fistula. Right. Possibility of breaking the wall of the abscess between two loops of bowels with production of fatal general peritonitis

may fluctuate. Then it is time to open and drain the abscess. It may point to the anterior abdominal wall and then is opened by incising that wall. If it gravitates to Poupart's ligament, it may be opened retroperitoneally by an incision parallel to that ligament. If the abscess is formed in the pelvis, it will gravitate downward and may rupture either through the anterior rectal wall or through the vagina. If the abscess descends but does not open spontaneously, it should be opened by incising the anterior rectal wall after the presence of pus has been ascertained by a needle inserted into the anterior wall of the rectum; a bistouri is inserted along the needle. As soon as the opening is made through the anterior wall of the rectum an artery forceps is introduced, the blades are opened, pus is evacuated and a small Penrose drain is introduced.

If the abscess is localized in the right iliac fossa, it should be opened at the point of the highest protrusion. Extreme care should be taken not

to enter the general peritoneal cavity while approaching the abscess or later by breaking the abscess from inside (Fig. 182). When the abscess is opened, one should not search for the appendix. If the appendix presents itself and is easily delivered, it may be removed, otherwise a few Penrose drains or two medium size rubber tubes should be inserted (Fig. 183). We stress particularly the danger of making a rough digital examination of the abscess-cavity on account of the possibilities either of breaking through the wall of a loop of bowel, which served as a wall of the abscess cavity, thus producing a fecal fistula or, what is even worse, of breaking the wall of the abscess cavity between two loops of bowel, thus entering the free peritoneal cavity and producing peritonitis (Fig. 184). We advise a bacteriologic examination of the pus, since it has a fairly reliable prognostic significance. If the pus contains *Bacillus coli*, the prognosis is fair; if it contains streptococci, the prognosis is worse; and if *Bacillus Welchii* are found, the condition is grave. The operative procedure should be followed by administration of sulfa drugs or penicillin.

Appendicitis in Children

Acute appendicitis in children is less frequent than in adults. When speaking of children, we mean people up to thirteen years of age. It is very rare under the age of two. Griffith, in 1901, was able to collect only fifteen cases from the literature and Abt, in 1917, eighty cases under the age of two. From the age of five it increases considerably and this increase is noted with each additional year, reaching the maximum at the age of thirteen. We do not wish to imply that after the age of thirteen it diminishes in frequency, which is not the case: but after this age it belongs to the group of adults. A review of large statistics shows that the incidence of acute appendicitis up to five years of age comprises about 10 per cent of all cases in children and from five to thirteen years 90 per cent. In comparison with adults, statistics reveal that children from one to five years of age constitute slightly above 1 per cent of all cases of appendicitis and from five to ten years, 4.3 per cent.

SYMPTOMATOLOGY. After the age of five, the symptoms are essentially the same as in adults. Pain, nausea and vomiting, tenderness, rigidity, elevation of temperature and leukocytosis are manifested along the same lines as in adults. However, appendicitis in children is a more serious problem than in adults for the following reasons:

It is difficult to elicit a good history, physical examination is more difficult and very often cathartics are given by parents before they visit the doctor. In addition to these difficulties there are definite anatomophysiology drawbacks: The wall of the appendix is very thin and is liable to perforate more readily than in adults; the blood supply is inadequate, thus enhancing gangrene of the appendix; the omentum is

short and thin and, therefore, is not as useful for "walling off" the inflamed area from the rest of the peritoneal cavity as in adults. All these factors explain the high mortality in children. A statistical survey made of a large number of cases shows that the mortality rate among children below two years of age is about 25 per cent, from three to five years 15 per cent, from six to ten years 8 per cent and for the entire group it is about 10 per cent. Diagnosis in the majority of cases is easy, yet at times it presents insurmountable difficulties. One has to exclude acute infectious diseases, notably scarlet fever and measles, which may manifest themselves by vomitus, abdominal pain, rigidity, elevated temperature and leukocytosis.

PNEUMONIA, PYELITIS AND ACUTE GASTROENTERITIS from time to time are diagnosed as acute appendicitis. Intussusception occasionally is mistaken for acute appendicitis with "mass-formation."

ACUTE MESENTERIC LYMPHADENITIS has also been confused with acute appendicitis.

The symptoms offer a striking resemblance to appendicitis: pain, tenderness, rigidity, mostly in the right lower quadrant, slight fever, leukocytosis of 15,000 to 20,000 with increased polymorphonuclears. Occasionally, a differential diagnosis can be made before the operation, namely, under an anesthetic a chain of lymphatic glands can be palpated. Only after the abdomen has been opened does one find a normal or slightly inflamed appendix with a chain of enlarged lymphatic glands.

Treatment is essentially the same as in adults, with this probable difference that the indication for entering the abdomen as soon as the child is seen, irrespective of the length of time elapsed after the onset, is more imperative than in adults in whom the time limitation for operative intervention should be adhered to more strictly. However, if a mass is palpated in the abdomen, conservative treatment should be employed. As children stand operative shock and hemorrhage poorly, it is important to prepare them preoperatively. The intravenous infusion of saline and glucose before and after operation is very important.

Appendicitis in the Aged

We consider all cases of appendicitis occurring in people after fifty as appendicitis of the aged. The older the age group, the less frequent is the incidence of appendicitis and the higher is the mortality. Thus, between the ages of fifty and sixty appendicitis occurs in about 2.7 per cent and between sixty and seventy in about 0.7 per cent. It is more serious than in adults or in children. There is a male preponderance of almost 2:1 over the female and the mortality ratio is even larger. The mortality rate is considerably higher for the group between fifty and sixty years reaching about 15 per cent, and 30 per cent for the group between sixty and seventy.

There are several reasons which account for this high mortality. To begin with in old people appendicitis is mostly of the vascular type and has not an infectious or occluding the lumen of appendix origin as is usually the case in young people. It is the occlusion of the appendicular artery which causes appendicitis. As a sequela of vascular occlusion, massive gangrene of the entire appendix occurs and it takes place in a very short time. Only after this occurs do inflammatory symptoms appear, which force the patient to seek medical aid. In about 75 per cent of all cases the appendix is gangrenous within twenty-four to forty-eight hours after the onset, it is of the massive variety and about 30 per cent of them have already perforated. As the origin is mostly vascular the tendency to produce occlusion along the veins is great. Very often on the operating table one sees the spreading of the inflammation along the ileocecal vein; in more extensive cases, of the superior mesenteric vein, and in some cases of the vena porta-pylephlebitis with single or, more frequently, multiple abscesses of the liver for which Dieulafoy long ago coined the name of "appendicular liver."

SYMPTOMATOLOGY. The symptoms are not as characteristic as they are in younger adults and the pathological picture in the first seventy-two hours is far graver than the clinical picture. Pain may be insignificant and may not localize for days, if ever; vomiting may be absent and it may precede pain; the temperature may be normal or even subnormal. Rigidity is present in about 40 per cent, Blumberg's sign in about 50 per cent and Rovsing's sign in 40 per cent. Such patients do not consider themselves very ill and often will have taken cathartics once or repeatedly before seeing a doctor. The leukocyte count in the average case is less than 15,000 and polymorphonuclear cells are less than 80. There is a general opinion that ability to form adhesions is poor in old people. We think that this is not the case; we believe on the contrary that it is good, but the process of "walling off" does not take place as frequently as in adults due to the rapidity of gangrene development so that there is not enough time for "walling off." Therefore, the diffuse peritonitis is present in about 15 per cent of the aged operated upon within forty-eight hours after the onset. Here, as in any other group, the close relationship between the mortality and the time of the operation is clearly seen. While the mortality for all cases of appendicitis is about 2.9 per cent if operated upon within twenty-four hours after the onset, it is about 10.5 per cent for the group above forty, and while the mortality is 4.6 per cent for the entire group operated after forty-eight hours, it is about 13 per cent for the group of over forty.

The French authors for many years divided appendicitis of the aged into four groups: catarrhal, gangrenous, pseudoneoplastic and pseudo-occlusive; in other words, it occurs as catarrhal or gangrenous appendicitis, or may simulate neoplastic disease of the cecum or intestinal

obstruction. The reasons for the pseudo-neoplastic and pseudo-occluding varieties are simple: Infiltration or mass-formation due to appendicitis has already taken place before the patient seeks medical aid, typical symptoms of appendicitis are absent and the surgeon, feeling a mass, certainly thinks of carcinoma, or hearing of difficulty in passing gas thinks of intestinal occlusion. Among the erroneous diagnoses made (and these occur in old age in about 40 per cent) are biliary colic, renal colic, carcinoma of the large bowel and intestinal obstruction. The latter two diagnoses are made particularly often because the patient applies for aid in the stage of infiltration when acute symptoms, if they have been present, have already disappeared.

The most frequent cause of death in old people is peritonitis, then pneumonia, cardiac and cardiorenal disease, pyelephlebitis, toxemia, paralytic and mechanical ileus, and cerebral hemorrhage.

TREATMENT. If early operation is important in any age, it is particularly important in old age, because the pathologic change develops extremely fast due to vascular occlusion, and gangrene of the appendix with perforation occurs early. If a mass is palpated, conservative treatment should be employed.

In the presence of local or of diffuse peritonitis the management is the same as discussed under the titles: "Management of Spreading Peritonitis" and "Management of Diffuse Peritonitis."

Appendicitis in Hernia

Acute inflammation of an appendix situated in a hernial sac presents different clinical pictures and for this reason the diagnosis is extremely difficult. Suffice it to say that nearly all patients with acute appendicitis in a hernial sac were operated upon because of a diagnosis of strangulated hernia and the finding of an acutely inflamed appendix was rather an unexpected surprise. This incidence does not occur often, approximately in about one-half of 1 per cent of all cases of strangulated herniae. The appendix is found most frequently in the right inguinal hernia and less frequently in the right femoral hernia. However, cases have been reported in which the appendix was found in the left inguinal and in the left femoral hernia, evidently when the cecum has been extremely mobile. The appendix may rest in a hernial sac for years without giving any symptoms. It may become strangulated in a hernial sac together with the rest of the hernial contents. The appendix may not be strangulated, while other contents of the sac are, and again the appendix may be the sole contents of the hernia and become strangulated.

Therefore, since the presence of the appendix in the hernial sac is possible, one should remember this if the previous history states repeated pain of short duration in the hernia, which is accompanied by moderate elevation of temperature and no strangulation.

Physical findings will show elevated temperature, occasionally edema and redness of the scrotum, soft, not distended abdomen and pain in the entire hernial mass, whereas with a strangulated hernia there will be symptoms of obstruction, pain only in the hernial ring, pain radiating to the umbilicus and normal temperature, at least during the first few hours. The dangers connected with this type of hernia are evident, since the surgeon may try to reduce it and by manual applications may rupture the appendix.

The treatment consists of appendectomy and herniotomy.

Retrocecal Appendicitis

The question of retrocecal appendicitis does not attract as much attention as it deserves. There is not only lack of unanimity in the presentation of the clinical picture but even in the definition of a retrocecal appendix. Some consider the appendix to be retrocecal if the proximal portion of it is behind the cecum, irrespective of the position of its distal portion; others consider the appendix to be retrocecal only in those cases in which it lies in its entirety behind the cecum, irrespective of whether the cecum is movable or immobile; still others consider it retrocecal only if it lies behind the immobile cecum and colon and in order to expose it, it becomes necessary to mobilize the cecum. Depending on the interpretation of the term retrocecal, statistics differ as to the frequency of its occurrence. This will also account for the different description of symptomatology as given by different investigators. It is evident, that in those cases in which the appendix is considered retrocecal when only its proximal portion lies behind the cecum, the symptoms will not differ from cases of appendicitis of the non-retrocecal type, whereas the symptoms will be different when the appendix lies in toto behind an immobile cecum or colon ascendens. In our own statistics published in 1932 relative to the frequency of its occurrence we considered an appendix retrocecal if it lay in toto behind the cecum or colon, so that it was not seen until the cecum was misplaced; however, we did not consider it necessary that the cecum be immobile, so that for exposure of the appendix it would not be necessary to mobilize the cecum or colon. In other words, we consider the appendix retrocecal as long as it lay in its entire length behind the cecum or colon, irrespective of whether it was intraperitoneal or retroperitoneal. With this definition we found a retrocecal appendix present in 9 per cent of all cases.

Clinically, retrocecal appendicitis is characterized by grave symptoms due to great pathologo-anatomical changes, by the variety of complications, technical difficulties met with in the removal of the appendix and by the more serious postoperative course.

The location of the appendix in the retroperitoneal space (if it happens to be this type), plastering to the wall of the cecum, and close proximity

of the ureter, kidney or liver, clearly explain the gravity of complications and the resemblance of some other diseases. As a rule, the blood supply to the appendix in a retrocecal position is poorly developed; this is the reason why so often retrocecal appendicitis is accompanied by gangrene and perforations.

SYMPTOMATOLOGY. In retrocecal appendicitis one may encounter all the symptoms characteristic of inflammation of a normally situated appendix; however, they are not as constant and not as pronounced. Pain, nausea and vomitus, tenderness and rigidity are less pronounced but in most cases are present. The pulse and temperature do not show any deviations from cases of inflammation of normally situated appendices. However, retrocecal appendicitis occasionally presents a few additional symptoms not found in normal appendicular cases. There may be pain in the lumbar region between the twelfth rib and the crest of the ilium just lateral to the musculus erector trunci; occasionally there is tenderness over the trigonum of Petit; sometimes there is pain in case of over-extension of the right thigh. Pain in micturition and frequency of urination are observed from time to time. Occasionally the patients complain of radiating pain into the right lower extremity. V. T. Williams reported a sign which may be valuable in the diagnosis of acute retrocecal appendicitis, namely, if the patient is placed in a prone position, the tenderness in the lumbar region decreases and in the right lower quadrant it increases, as contrasted with the tenderness experienced when the patient lies on his back.

Another sign which proved to be of great value in making a diagnosis of retrocecal appendicitis is the sign of Kerangal: If the patient is placed on the left side and palpation is applied to the right iliac fossa, there will be an increase in pain, which otherwise is either slight or altogether absent. This is explained as being due to the dropping of the cecum forward and downward, thus exposing the appendix which is then directly compressed by palpation. However, this sign will not work when the cecum is immobile, which occurs in 20 per cent of all cases.

As this form of appendicitis is accompanied more frequently by gangrene and perforations, immediate operation is indicated even more than in inflammation of normally placed appendices.

TECHNIC. The incision should be of the gridiron type but placed closer to the anterior superior iliac spine than is usually done. If after opening the abdomen the diagnosis of a retrocecal appendix is confirmed, the incision is enlarged (if it was not adequately large) by prolonging it upward and lateralward; the cecum is mobilized if it is a case of an immobile cecum, or just turned medialward if it is a mobile cecum. The next step depends on whether the appendix lies on the posterior abdominal wall or on the posterior cecal wall. If it lies on the posterior abdominal wall, the technic of removal is simple; if it is plastered against

easily break the wall of the cecum and enter that organ. The technique of removal when the appendix is plastered against the posterior cecal wall is as follows: The visceral peritoneum is cut on the wall of the cecum parallel to the appendix for a distance of half an inch; a curved forceps is introduced through this slit behind the appendix and between it and the wall of the cecum and the appendix is then separated from the cecal wall; then the visceral peritoneum, which binds down the appendix to the wall of the cecum, appears in marked relief and can easily be cut from the cecum without the danger of injuring its wall.

DRAINAGE in the intraperitoneal type of appendicitis follows the same principles as those in the normal type. However, if the appendix lies retroperitoneally and is gangrenous, it is better to close the posterior parietal peritoneum and pass a Penrose drain through a lumbar incision.

Among the complications which arise in this position of the appendix (and they arise more frequently than when the appendix is in a normal position) are abscess of the Douglas pouch, ileus, paranephritis, pneumonia, pleurisy and pyelophlebitis. The mortality rate is about twice as great as that for appendicitis with the appendix in a normal position.

Appendicitis in Pregnancy

Acute appendicitis in pregnancy is usually seen in the early months. It is considerably rare in the late months of pregnancy; however, cases immediately preceding labor and also in puerperium have been reported from time to time. Acute appendicitis developing during labor is also rare. Records show that more than 80 per cent of all cases of appendicitis in pregnancy are seen before the sixth month.

This condition is very dangerous for many reasons. To begin with, it is more difficult to make an early diagnosis. A physician is apt to consider that all the symptoms are due to disturbances in pregnancy, and when there is a suspicion of appendicitis, there is a tendency to delay the operation. If, finally, the operation is decided upon, technical difficulties of appendectomy arise. All these factors, namely, difficult diagnosis, delay in operative intervention and technical difficulties increase the mortality. Some surgeons who have had experience with this type of case claim that acute appendicitis in pregnancy progresses rapidly and has a tendency toward early perforation, and for this reason the patient should be operated upon immediately after the diagnosis is made.

When the appendix perforates and forms an abscess, the gravid uterus usually forms one of its walls. If miscarriage takes place, the uterine contraction facilitates the rupture of the abscess with ensuing general peritonitis. The incision should be the gridiron type but made larger and higher than the typical McBurney incision.

Left-sided Appendicitis

This occurrence is comparatively rare. Up to 1935 seventy-three cases of this type were reported. In about two-thirds of this group (forty-eight cases) the entire appendix was situated on the left side. Of this group of forty-eight cases, thirty-four showed complete transposition of the viscera (*situs viscerum inversus totalis*, that is, all structures occupied a transposed position as in a mirror, the right liver lobe lay to the left of the falciform ligament, and the left liver lobe to the right of the same ligament; the gallbladder lay to the left of the falciform ligament, the stomach to the right of the spinal column, the pylorus was directed to the left, the pyloroduodenal junction lay to the left of the spinal column, and the cecum and appendix were in the left iliac fossa.) In twelve, only a partial transposition of the viscera occurred (*situs viscerum inversus partialis*, in which only some of the structures occupied the "mirror" position). Two cases were of the *mesenterium commune* type in which all small bowels lay to the right of the spinal column and all the large bowels to the left.

However, in one-third of all cases in which the appendix occupied a left-sided position, there was no *situs inversus* but pathological processes pushed the cecum and the appendix to the left so that both of them or only the appendix reached the left iliac fossa. Occasionally, the cecum will lie in the normal position but the appendix is very long, bridges over the pelvis and reaches the left iliac fossa.

The clinical picture varies greatly. In total transposition of the viscera the pain, tenderness and rigidity may be on the left side. However, when the appendix is on the left side, but its base is on the right side where the cecum lies, the pain, tenderness and rigidity may be bilateral with predominant pain on the right side. The diagnosis of a left-sided appendicitis is extremely difficult which is attested to by the fact that in more than 90 per cent of left-sided appendicitis the abdominal incision was made on the right side and it was necessary to make another incision on the left.

Symptomatic Appendicitis in Supposedly Congenital Absence of the Appendix

Cases have been reported in which the patient presented typical symptoms of appendicitis in which a laparotomy was performed and no appendix was found. The abdomen was closed and later the patient again had attacks of appendicitis. We firmly believe that these were not cases of congenital absence of the appendix but a rare type in which the appendix lay behind the visceral serosa of the wall of the cecum or, in other words, the surgeons who met such cases were dealing with a truly

room, but came across such a condition twice on the dissecting table. In both these cases there was an immobile cecum and a Lane kind present. The presence of a Lane kink enabled us to decide beforehand that we were dealing with a retrocecal appendix and that it was necessary to mobilize the cecum in order to see the appendix. After mobilization of the cecum (Fig. 170) no appendix was seen or felt. Incising the serosa on the posterior wall of the cecum at the level of the ileocecal junction about one-half an inch from it, we found in both of these cases an appendix normal in length and diameter, curled in the form of a semicircle and of a normal consistency (Fig. 172). We firmly believe that the same position of the appendix can account for a definite number of reports of a "congenital absence of the appendix" not only not found on the operating table, but even for cases not found on the dissecting table. We presume that at least some of the specimens carefully preserved in museums as specimens of a cecum with an absent appendix have a normal appendix completely covered by the serosa of the cecum.

Chemotherapy and Serotherapy in Appendicitis

Serotherapy consists of the subcutaneous or intramuscular injection of different serums. It should be understood that this is not done to replace surgery but is done only when surgery is already contraindicated as for instance when mass formation is present or general peritonitis has developed. Many surgeons in France and some of us in this country have successfully used Weinberg's antiperitonitic serum. This is a combination of the polyvalent antigangrenous serum, anticolibacillus serum and serum of other bacilli, such as enterococci, streptococci, *Bacillus ramosus*, *Bacillus funduliformis*, *Bacillus fusiformis*, *Staphylococcus parvulus*, etc. The serum is injected subcutaneously or intramuscularly. The amount of serum used is about 80 to 100 cc. for each injection. This portion is diluted in 250 cc. of normal salt solution and the entire amount is injected. A second dose of the same amount is injected twenty-four hours later and the third doses in the following twenty-four hours. However, with advent of chemotherapy (sulfa drugs), and penicillin, serotherapy is not much in use. Sulfa drugs and penicillin are used not only when surgery is contraindicated, but also as supplement to surgery.

Traumatic Appendicitis

By traumatic appendicitis is meant inflammation of the appendix due to injury by some external force. There is no unanimity among the students on the subject as to whether or not traumatic appendicitis exists as a clinical entity. Three opinions on the matter are prevalent: One considers that there is no such entity as traumatic appendicitis and re-

gards occasional attacks of appendicitis following an injury as a mere coincidence. Another opinion is that the injury exacerbates a previously dormant, inflamed appendix but denies the possibility that an injury may produce inflammation of an appendix otherwise normal. The third opinion is that it is possible to produce appendicitis by an outside force, and this opinion is gaining more and more recognition.

The trauma may be direct, such as a blow to the abdomen, a kick or a fall on the abdomen, pinioning it against a fixed object. Indirect trauma is produced by a strong action either of the muscles of the anterior abdominal wall or of the psoas major muscle.

While the mechanism of wounding the appendix by a direct force is clear, although it is very infrequent, the mechanism of an *indirect* trauma is not clear. The most accepted theory of the pathogenesis of traumatic appendicitis by an indirect force is that this force compresses the cecum and forces its contents into the appendix. This forced contents may break and injure the mucosa of the appendix and serve as a starting point of appendicitis; if the appendix has a normal sized lumen, it will free itself from this contents by emptying them back into the cecum. If, however, chronic pathologic conditions are present, such as angulating adhesions, scars and particularly coproliths, the contents cannot evacuate; there is defective drainage of the contents, bacterial growths take place and acute appendicitis develops. Indeed, in many cases of traumatic appendicitis fecaliths are found at the time of operation.

Howard Kelly, in his classical book, "The Vermiform Appendix and its Diseases," recorded fifty cases of traumatic appendicitis collected from the literature. Out of 1,260 cases of acute appendicitis treated in Charity Hospital in New Orleans over a three year period Urban Maes was able to find only six cases of traumatic appendicitis or less than one-half of 1 per cent. However, some surgeons believe the incidence of traumatic appendicitis to be considerably higher, namely, up to 5 per cent, and a great number of cases have been reported here and abroad.

The clinical picture of traumatic appendicitis is the same as that of a non-traumatic origin, the only difference being that it starts immediately after a force has been applied to the abdomen directly or indirectly. The diagnosis and treatment are of course the same as in any other case of acute appendicitis.

Comparative Data of Incidence, Course and Prognosis of Acute Appendicitis in Two Sexes

Whether there is a preponderance of incidence in either sex is debatable. In the statistics of old authors the preponderance of the female over the male is considerable. Later, other statistics were published showing a male preponderance over the female at the ratio of 60 to 40.

Recently, some statistics attempted to show that there is no difference in incidence in either of the sexes.

As far as gravity of the course is concerned, numerous statistics show that up to the age of fifteen and after the age of forty the gravity and mortality are about the same in both sexes; but between the ages of fifteen and forty the course is graver and the mortality is higher in men, the death rate being about three times as high as that in the female (3 per cent and 1.12 per cent, respectively). This cannot be explained by the fact that light cases occur more frequently in women than in men, because cases of equal gravity also produce a higher mortality in men. There is a tendency to explain this by the greater defensive power in women whose pelvic peritoneum has cyclic hyperemia in the time of menstruation and also greater acquired immunity to infection due to the constant communication of the peritoneal cavity with the outside world through the tubes, uterus and vagina.

The process of "walling off" the pathological appendix from the rest of the peritoneal cavity is ten times more frequent in women than in men, that is, formation of an infiltrate or of a "mass formation." This, of course, diminishes the chances of general peritonitis. In pelvic appendicitis, the uterus and adnexa often serve in "walling off" the abscess.

WHEN TO RISE

The question of early rising was discussed by us in the chapter on laparotomy to which we refer the reader. In appendicitis, if the patient has been operated upon under local anesthesia and a McBurney incision has been made, he may rise from the bed within the first twenty-four hours. As a matter of fact a great number of cases have been reported in which the patients were allowed to walk directly from the operating table if the operation was performed under local anesthesia, or in twenty-four hours if it was done under spinal anesthesia. In those cases in which the abdomen is drained the patient remains in bed for eight days or more depending on the progress of the disease.

Preoperative and Postoperative Management in Appendicitis

Preoperative care is very simple. Morphine with atropine is given as a basic anesthetic preliminary to general anesthesia, or morphine without atropine in case of spinal or local anesthesia. No enema should be given preliminary to the operation.

Postoperative management varies much more than preoperative, depending on the type of pathology found at the time of operation and the complications which develop postoperatively. In uncomplicated appendicitis we give the patient from 2,000 to 3,000 cc. of normal salt solution as hypodermoclysis or venoclysis. In some patients, as indicated below, we add 5 per cent glucose in case of hypodermoclysis or 10 per

cent glucose if given as venoclysis. Nothing is given orally for the first twelve hours and only sips of warm water in the second twelve hours. After twenty-four hours we allow two ounces of liquid every two hours and keep giving 2,000 cc. of normal salt solution with or without glucose hypodermically or as venoclysis during the second twenty-four hours. On the third postoperative day the patient is given four ounces of liquids every three hours and the hypodermoclysis is discontinued. On the fourth postoperative day the patient is placed on a soft diet. This management is carried out when we keep the patient in bed for several days after the operation. If, however, we allow the patient to rise twelve or twenty-four hours after operation, we discontinue the hypodermoclysis before the patient leaves the bed.

Sedatives are given during the first and second days to diminish pain and a rubber tube is inserted in the rectum in case of gas pain.

On the second day the patient is permitted to sit in bed and on the third on a chair, provided that the abdomen was opened by McBurney incision. Stitches are removed on the ninth day.

However, when complications arise such as sepsis, peritonitis, ileus, abscess or prostration, these require special attention. Penicillin and sulfa drugs are given. Sulfa is given up to four grams daily and penicillin up to 300,000 units daily. Dehydration has to be fought against and fluid balance preserved. The intravenous or hypodermic injection of fluid is of great value. In normally progressing cases about 2,500 cc. of water is lost by respiration, evaporation and excretion of urine, but those who have high fever, rapid respiration and perspire freely lose considerably more. This amount should be replaced and in addition more should be added to make up for the lack of fluid in patients already dehydrated. This is the reason, why the amount of fluid injected may reach up to 3,000 cc. In cases in which a large amount of fluid has been injected it is important to study the degree of concentration and the chloride level of the circulating blood in order not to overload the circulatory system. Examination of the urine in regard to specific gravity, presence of acetone and diacetic acid will indicate whether more salt or glucose is required and when an adequate amount of fluid has been furnished.

DISTENTION of bowels should be combated. If it is moderate, abdominal poultices are sufficient and in case of gas pain, a rectal tube is inserted. In more pronounced cases prostigmine should be given—1 ampule every two hours until distention disappears. In **paralytic ileus** suction through a nasal catheter should be employed. The finding of considerable peritoneal infection at the time of operation is a positive indication for starting continuous suction drainage.

REST is of the greatest importance to the patient and *no medication* should be given to the patient while he is asleep.

POSITION. The horizontal position should be employed while the patient is in a state of anesthesia and after that Fowler's position, particularly if there is diffuse peritonitis.

Mortality in Appendicitis

The frequency of death from appendicitis depends on a great many factors. Some of them are constitutional, such as the general resistance of each person afflicted, age, etc. Other factors are the severity of infection, the time elapsed between the onset of the disease and the time of operation, the taking of cathartics, etc. All of these greatly contribute toward mortality but two are most important: the time elapsed between the operation and the onset, and whether the patient has taken cathartics. The influence that the time of operation has on the mortality is seen from the following table:

Time of Operation	Mortality Per cent
Within 24 hours	2.06
Between 24-48 hours	4.88
Between 48-72 hours	6.67
After 72 hours	9.70

In other words, the possibilities for death are twice as great if the patient is operated upon the second day, three times if on the third day and five times if on the fourth day as compared with those operated upon the first day. That the taking of cathartics increases the chance of perforation is attested to by many surgeons.

AGE. The older the patients the higher is the mortality, but as the number of patients afflicted with this disease decreases with advancement in age, the total number of deaths in old age is not great. Eighty per cent of all fatal cases occur between the ages of fifteen and forty.

POSTOPERATIVE MORTALITY varies in different countries. In the United States it is about 5.74 per cent, in Europe 5.10 per cent.

Causes of death in their order of frequency are: General peritonitis, circumscribed peritonitis, general septic infection, pneumonia and pleurisy, liver abscess, thrombosis of the mesenteric or portal vein and subdiaphragmatic abscess. Since the advent of sulfa drugs and penicillin, the mortality in appendicitis considerably decreased.

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Chapter XIII

Intestinal Obstruction

By JOHN R. PAINE

INTRODUCTION

During the past ten years there has been a distinct change in the attitude of both surgeons and internists toward the problem of intestinal obstruction. The two main factors which have produced this changed attitude are: (1) the proper appreciation of the fluid-electrolyte disturbance which accompanies obstructions of the small bowel, and (2) the elucidation of those mechanical factors which can be controlled by properly employed surgical procedures.

No longer does the diagnosis of intestinal obstruction mean necessarily an emergency laparotomy. Moreover, no longer can the surgeon be satisfied with the mere diagnosis of "obstruction." Modern methods of treatment demand that the approximate site and degree of obstruction, and whether or not this obstruction is of a strangulating character, be determined. On the basis of this knowledge, the surgeon must then decide whether the best interests of the patient require an immediate operation, an operation of election after a day or two, or an attempt at conservative treatment. Thus, with increased knowledge has come increased responsibilities. That these responsibilities are being met, at least in part, is indicated by the reports of decreased mortalities in relatively large series of cases which have begun to appear in the current medical literature. This apparent decrease in mortality is the first evidence of improvement in the treatment of this condition since the advent of aseptic surgical methods.

CLASSIFICATION. Obstructions of the bowel may be classified in a number of ways. It is singularly important for purposes of treatment and prognosis to determine whether an obstruction is **complete** or **partial**. By a complete obstruction is meant an obstruction of the lumen of the bowel which completely prevents the passage of any gas, fluid or solid beyond it. A partial obstruction is represented by a narrowing of the lumen of the bowel so that its contents pass only with difficulty and after some delay at the site of the obstruction. Not infrequently a partial obstruction suddenly becomes complete due to the impaction of the fecal content or to edema of the bowel wall.

It is extremely important to distinguish between a **simple** obstruction and a **strangulated** obstruction. In the broadest sense simple obstruc-

tions include those mechanical obstructions in which the blood supply to the bowel is not compromised as well as those obstructions due to nervous dysfunction of the musculature of the bowel wall. There may or may not, therefore, be an actual occlusion of the lumen of the bowel. The term, strangulation, connotes some obstruction to the blood flow through a segment of the intestine. The obstruction to blood flow may be

TABLE I
A Clinical and Pathological Classification of Intestinal Obstructions
(after Wangensteen)

Clinical Classification	Pathological Classification
I. Mechanical	
A. Narrowing of lumen	
1. Strictures of bowel wall	
a. Congenital: Atresia	
Imperforate anus	
b. Acquired: Inflammatory	
Traumatic	Simple except in neoplastic strictures of the colon
Vascular	
Neoplastic	
2. Obturation	
3. Compression from without, especially pelvis and retroperitoneal duodenum	
B. Adhesive bands	
1. Congenital	Simple or strangulated
2. Inflammatory	
3. Traumatic	
4. Neoplastic	
C. Hernia	
1. External	Strangulated
2. Internal	
D. Volvulus	
E. Intussusception	
F. Errors in development of the intestine	
II. Nervous (physiological)	
A. Inhibition ileus—(paralytic)	Simple
dynamic ileus	
B. Spastic ileus—dynamic ileus	
III. Vascular	
A. Thrombosis of mesenteric vessels	Strangulated
B. Embolism of mesenteric vessels	

in the arteries or veins, or in both. In addition there may or may not be an actual occlusion of the bowel lumen. In either event the transport of the bowel contents past this involved segment of bowel is interrupted. The obstruction may be due entirely to an absence of the normal peristaltic movements in the infarcted segment of the intestine. The presence of an infarcted segment of bowel is of far greater immediate importance than the presence of an obstruction *per se*. Whether the infarction be due to an occlusion of the veins or arteries is also of prognostic and

therapeutic importance, but such a distinction can not usually be made with any degree of certainty before operation.

Most discussions of intestinal obstructions concern themselves with a clinical classification. Such a classification is anatomic in scope and indicates the various types of lesions producing the obstruction. Examples of such a classification are given in Table I.

A comprehensive clinical and pathological classification has been given by Wangenstein and is repeated in Table I. This classification will be followed in general in the succeeding sections of this chapter.

TABLE II
Relative Incidence of Anatomic Causes of Intestinal Obstruction

	Scudder, Zwemer and Whipple: Presbyterian Hospital, New York	Vidgoff: Los An- geles County General Hospital, 1925-1930	Cornell: New York Hospital, 1913-1930
Total Cases	2,150	264	235
Hernia: External	1,000	49	10
Internal	36		
Adhesions and bands	573	170	110
Volvulus			16
Small intestine	156	0	
Large intestine	127	4	
Intussusception	69	11	36
Gallstones and foreign bodies	17	4	4
Meckel's diverticulum	15	2	7
Mesenteric thrombosis	35	0	0
Miscellaneous	36	0	36
Cause not determined	86	0	0
Cancer	not included in series	22	16(colon)
Diverticulitis	not listed separately	2	not listed separately

INCIDENCE. McCormick, in 1930, reported the results of a questionnaire sent to twenty-two hospitals of one-hundred beds or more in the states of Indiana, Ohio and Michigan. He found that 1.93 per cent of 61,401 abdominal operations were performed for intestinal obstruction; 1.40 per cent were performed for primary obstruction; and .53 per cent were performed for postoperative obstruction. McIver reported, in 1932, that .83 per cent of 39,936 surgical admissions to the Massachusetts General Hospital over a ten-year period were for intestinal obstruction. These figures give a fair estimate of the numerical importance of intestinal obstruction as contrasted with other surgical conditions seen in the United States.

The relative frequency of various types of obstruction varies a great

deal in different parts of the world. Intussusception formed 45 per cent of 342 cases of acute intestinal obstruction recently reported by Todyo from Japan. In Eastern Europe the incidence of volvulus is well known to be greater than elsewhere. Perlman, in Russia, found that over one-half of 215 cases of obstruction which he reviewed were due to this cause. In addition to these geographical variations others are found as existing between different surgeons and hospitals depending upon the character of the patients whom they serve. The relative frequency of the various anatomic types of obstruction in the United States can best be appreciated by the comparison of large reported series such as are given in Table II. Hernia and adhesive bands are by far the most frequent causes of intestinal obstruction in this country.

The influence of previous abdominal surgery in the etiology of obstruction has been pointed out frequently. Vidgoff found that 68 per cent of the patients in his series had had previous abdominal operations. Other series such as Finney's and Miller's showed that 50 per cent and 24.2 per cent of patients, respectively, had had a previous laparotomy. These figures are reflected in the relatively high incidence of adhesive bands as a cause of obstruction.

PATHOLOGICAL PHYSIOLOGY. The fundamental disturbance of the physiological activities of the body incident to intestinal obstruction is the stasis of fluid, feces and gas in that portion of the alimentary canal proximal to the site of the obstruction and the consequent development of distention. As a secondary effect the fluid and electrolyte balance of the body is altered. If the obstruction be long continued, other deleterious effects are produced such as increase in the bacterial population of the bowel contents and increased permeability of the bowel wall. The extent and importance of these different factors vary with the site, character and degree of the obstruction.

In those instances in which strangulation of a segment of the bowel occurs simultaneously or as a sequel to the obstruction other consequences must be considered. These are (1) the loss of blood into and out of the strangulated segment and (2) the development of peritonitis due to the migration of bacteria through a necrotic bowel wall.

DISTENTION. The components of the distention in intestinal obstruction are: (1) gas, (2) fluid and (3) feces. The relative importance of these components is subject to wide variation. In certain instances, such as in partial obstruction of the colon, the accumulation of feces may account for the greater part of the distention. In complete obstruction, particularly of the small bowel, the accumulation of fluid and gas is the more important consideration.

GASEOUS DISTENTION. The gaseous content of the intestine in the presence or absence of obstruction is derived from the following sources: (a) swallowed air, (b) chemical reactions incident to digestion, (c) bac-

terial decomposition of organic substances in the intestinal chyme and (d) diffusion from the blood stream.

That portion of the gas derived from swallowed air is by far the largest. McIver, Benedict and Cline, as well as Hibbard and others, have indicated this fact and it is now generally admitted that approximately 70 per cent of all intestinal gas arises from this source. In the presence of obstruction, the portion of the gas due to bacterial action is increased and, due to stasis of the intestinal contents, there is an increase of the bacterial population. The relative amount of this gas, however, is small. The neutralization of the acid chyme of the stomach as it passes into the duodenum and upper portion of the small intestine normally produces a variable yet relatively large quantity of carbon dioxide. The importance of this in the production of the distention of obstruction must be questioned, however, because of the rapid diffusion of carbon dioxide through the mucous membrane of the intestine and because of the vomiting and reverse peristalsis which is usually present. Any collection of gas within the body tends to come into an equilibrium with the gases of the blood. Such an equilibrium involves the diffusion of gases into the blood as well as diffusion of gases out of the blood. It will, therefore, be apparent that the gaseous content of the intestine in the presence of obstruction is the resultant effect of many different factors operating simultaneously.

FLUID DISTENTION. Rowntree has estimated that about 7,000 ccm. of fluid in the form of digestive juices is normally secreted into the upper portion of the gastrointestinal tract each day. In the presence of obstruction of the small bowel the opportunity for the absorption of this fluid in the lower ileum and colon is prevented to a variable degree. This is particularly true if the obstruction be high in the small intestine. If the obstruction is in the large bowel, the absorption of fluid may be only slightly deranged. In any event, however, the fluid content of the bowel is usually increased in the presence of obstruction, and partakes directly in the distention of the proximal bowel. In addition to the fluid secreted into the intestine any fluid which the patient may swallow and retain after the onset of an obstruction also tends to increase the distention.

FECAL DISTENTION. Due to the rapid transit of chyme through the small intestine and because of its fluid content, the distention associated with obstructions of this portion of the gut is not greatly influenced by the mass of retained feces. In obstructions of the colon, however, and particularly in those complete obstructions which supervene on a slowly progressive partial obstruction and in certain neurogenic obstructions of the colon, retained feces may be a very important factor in the distention which develops.

EFFECTS OF DISTENTION IN OBSTRUCTION OF THE SMALL INTESTINE. Extreme degrees of distention produce a marked elevation of the dia-

phragm and may cause dyspnea and even cardiac embarrassment in some instances. The stomach is forced by the distention of the intestinal coils below it to assume a more horizontal position and is displaced to the right, sometimes quite appreciably. This fact must be borne in mind in the interpretation of roentgenograms of the abdomen taken for the purpose of diagnosis or to determine the position of indwelling suction tubes.

Of more direct importance are the effects of distention on the wall of the gut. In the presence of distention the small bowel through the contraction of its longitudinal muscle fibers shortens by approximately 25 per cent. At the same time the weight of the bowel wall is increased by about 33 per cent. This increase in weight is due to edema and is most noticeable directly above the point of obstruction.

The formation of this edema is the direct result of an increased pressure within the lumen of the obstructed bowel. Such a pressure tends to collapse the capillaries in the wall of the bowel and causes a filtration of fluid into the tissues. If this pressure is of sufficient degree and persists long enough, the capillary circulation is decreased to such an extent that actual necrosis of small areas on the antimesenteric border of the bowel occurs. Further effects of this nature are seen in those cases of simple obstruction which at operation are found to have relatively large quantities of free yellow opalescent fluid in the peritoneal cavity. At times this peritoneal fluid behaves as a mild irritant and leads to the mistaken diagnosis of probable strangulation. Also due to the edema, the tensile strength of the wall of the bowel is considerably decreased. This the author has noted at operation, and Sperling by actual measurement has found to be true. Furthermore, absorption from the lumen of the gut by way of the veins is decreased but absorption by way of the lymphatics is increased by distention.

The pressure within the lumen of the obstructed bowel is increased. Owings, McIntosh, Stone and Weinberg have found that while in normal dogs the average intraintestinal pressure is 2 to 4 cm. of water, the pressure in the obstructed small bowel is in the neighborhood of 8 cm. of water in the intervals between much higher pressures due to peristaltic movements. It has been determined by measurement in clinical cases at operation that the intraintestinal pressure in small bowel obstruction is usually 8 to 12 cm. of water. That such a degree of pressure, if long sustained, is capable of producing histological changes in the bowel wall is probable.

EFFECTS OF DISTENTION IN OBSTRUCTIONS OF THE COLON. In most people the ileocecal valve acts as an effective check against the retrograde passage of feces, fluid or gas, but permits these substances to pass into the colon from the ileum if sufficiently propelled by peristaltic

action. Therefore, in the presence of an obstruction of the colon a closed loop type of obstruction is formed with the distention which occurs, and its effects remain limited largely to the colon. Not infrequently, however, the action of the ileocecal valve is imperfect. In these cases the lower portion of the small bowel becomes distended in time and suffers the effects of this distention as have been described for small bowel obstructions.

In the typical case in which the ileocecal valve is competent, the intraluminal pressure may rise rather quickly to such a height as to obstruct the capillary blood flow in the walls of the colon and endanger its viability. Pressures consistently higher than those found in small bowel obstructions are seen in cases of obstruction of the colon. The intraluminal pressure may be over 23 cm. of water. This pressure exerts a constantly disrupting force on the walls of the colon which varies directly with the diameter of the bowel. For these reasons the frequency of perforation of the cecum in neglected obstruction of the colon and the necessity of an immediate decompressive operation in those cases in which the cecum is more than moderately distended, as seen on an x-ray film, is easily understood.

The general effects of distention on the colon have not been studied as completely as they have for the small bowel, but it is supposed that similar changes occur.

STRANGULATION. The presence of a strangulation obstruction produces the deranged physiology incident to a simple obstruction and in addition certain other phenomena which are peculiar to it alone. These latter effects are due to the infarction of the gut and must be thoroughly understood by any one who attempts to treat intestinal obstruction.

Strangulation is produced by some mechanism which obstructs the blood flow to a segment of bowel. In cases of mesenteric embolism the obstruction is within the arteries. In certain cases the veins may be obstructed by a thrombus. In most cases, however, such as those in which the obstruction is due to an adhesive band, an intussusception or a strangulated hernia, the vascular obstruction affects both the arteries and veins simultaneously. Since the veins are occluded by a lesser force than the arteries, however, the effect of the venous occlusion is felt first and overshadows any arterial occlusion. If the veins draining a segment of bowel be occluded, it rapidly becomes permeated with blood. The swelling is intense, the peritoneal surface loses its luster, bloody fluid fills the lumen of the bowel and filters out into the peritoneal cavity. If the segment of strangulated bowel is large, relatively huge quantities of blood are lost in this manner and shock rapidly supervenes. This in itself may cause death before time has elapsed for bacteria to migrate through the nonviable bowel and produce peritonitis. Thus a strangulat-

ing obstruction may cause death of either shock or peritonitis depending upon the size of the infarcted loop of bowel. In the presence of a purely arterial occlusion the loss of blood is much less. The immediate effects may not be as striking as those described above. With the passage of time, however, the bowel wall becomes permeable to bacteria and toxic substances which gain entrance to the peritoneal cavity and produce peritonitis and death. Intussusception frequently deceives the unsuspecting clinician because, in this instance, the infarcted bowel is completely encircled with viable tissue. The signs of peritoneal irritation considered characteristic of strangulation are usually entirely absent. Likewise in a strangulated hernia the findings are generally localized to the ensheathing and constricting hernial sac, the general peritoneal cavity being free of all signs of irritation.

DISTURBANCE IN THE FLUID AND ELECTROLYTE BALANCE. The importance of the loss of fluid and electrolytes in the vomitus and stagnant fluid in the intestinal tract of patients with obstruction was emphasized by Hayden and Orr, Hartwell and Hoguet. These losses are particularly severe in obstructions of the small bowel. In colonic obstructions they are minimal or may even be nonexistent. Hartwell and Hoguet further showed that in obstructions high up in the small intestine the dehydration and hypochloremia produced in this manner were the chief causes of death in untreated patients.

The effects of the dehydration produced in this way are the same as those seen from any other cause. The patient complains of thirst, the skin and subcutaneous tissues lose their turgor and the urine becomes scanty and highly concentrated. Due to an associated loss of electrolytes, effects other than those due to pure dehydration may appear. Although many electrolytes are lost in the vomitus and intestinal fluid, chloride is of chief importance. Persistent vomiting for thirty-six hours or longer will lower the blood chlorides to distinctly abnormal levels and produce a state of alkalosis. If such a condition persists, the patient may pass into the shock-like state of collapse associated with profound hypochloremia. In this condition the skin is frequently cold and moist. The lips and face are somewhat cyanotic. The pulse is weak and thready. The systolic blood pressure falls to 80 or 70 mm. of mercury and the diastolic pressure is unobtainable. A state of extrarenal uremia is produced as evidenced by an elevated blood urea nitrogen and nonprotein nitrogen.

The above discussion should indicate the importance of the para-oral administration of normal saline solution to the patient with obstruction in whom vomiting has been a prominent feature of the clinical picture. Within a few hours the deficiency in chlorides can be remedied and the prognosis of the patient greatly improved whether the obstruction *per se* be treated by operation or conservative methods.

Clinical Picture of Intestinal Obstruction

HISTORY. The taking of a careful history should be an essential part in the management of any case of intestinal obstruction. The story obtained should be carefully considered in all its component parts. It may serve only to confirm a diagnosis which seems obvious from physical findings, yet many cases occur in which the correct diagnosis can be made only after the sequence of events, as related by the patient, are appraised by the physician and integrated with the physical findings.

Particular importance should be paid to the following points in the history: (1) Age of the patient; (2) duration of the present illness; (3) character of onset of the symptoms; (4) type of abdominal pain and changes in the character of this pain since its onset; (5) frequency and severity of vomiting; (6) presence of audible intestinal noises; (7) frequency and character of bowel movements after the onset of symptoms; (8) decrease or absence of flatulence; (9) nature and extent of previous abdominal operations; and (10) recent loss of weight and strength.

SYMPTOMS: COMPLETE (ACUTE) OBSTRUCTION OF THE SMALL INTESTINE. The most striking symptoms of acute obstruction of the small bowel are **intermittent crampy pains and vomiting**. The onset of symptoms is sudden. Pain is usually referred to the midportion of the abdomen and experienced only at intervals. The pain is frequently described by the patient as like a "gas pain." For fifteen to twenty seconds it increases in severity and then gradually fades. The intervals between these pains may at first be only three or four minutes. With the passage of time, however, the interval becomes longer and the pains tend to become less severe. It would appear that these symptoms are directly related to the degree of distention of the bowel immediately above the point of obstruction. As the bowel becomes increasingly more distended peristalsis becomes weaker and less effective.

Vomiting at first is synchronized with the intestinal cramps but with the passage of time seems to occur at irregular intervals. In those cases in which the obstruction persists for more than a few hours the vomitus assumes a fecal character. Both its appearance and smell indicate that it comes from the small intestine. Initially the vomiting appears to be due to a nervous reflex but later takes on the appearance of an overflow phenomenon due to the regurgitation of intestinal contents into the stomach by reverse peristalsis.

The absence of defecation has long been regarded as one of the cardinal signs of intestinal obstruction. Patients are usually acutely aware of this change in their bowel habits. The physician must not be misled, however, by the occurrence of one or two small bowel movements after the onset of symptoms indicative of obstruction. Any fecal material present below the point of obstruction may pass on and be eliminated in the usual manner. It is unusual, however, for a patient to

have a defecation twenty-four hours after the onset of an obstruction unless that obstruction be partial or unless it has been overcome spontaneously.

The above remarks concerning the passing of feces in cases of obstruction also hold true for the passage of flatus.

COMPLETE (ACUTE) OBSTRUCTION OF THE COLON. Symptoms of acute obstruction of the colon are somewhat variable. Cramp-like pains recurring at considerably longer intervals than those noted in obstructions of the small bowel are usually present. The severity of these pains, however, may not be great and sometimes only close questioning will reveal that at the start of his illness the patient did have a few "gas pains." Vague, generalized abdominal distress is present in most cases. Vomiting is not a prominent symptom but may occur. The character of the vomitus is distinctly different from that seen in small bowel obstructions. It consists of small quantities of gastric and duodenal secretions. It would be rational to assume that in those cases in which the ileocecal valve is incompetent, vomiting should be more profuse and the vomitus of a fecal character. This may occur in rare instances but the author has not observed it.

Because the initial symptoms are not as severe as those present in small bowel obstructions these patients, as a rule, are seen somewhat later and the absence of flatulence and normal defecation may be striking. If the obstruction is in the sigmoid or left half of the colon the patients may notice an increase in the size of their abdomen and complain of the distention which produces this. On the other hand, obstructions in the right half of the colon may be accompanied by a distention sufficient to cause a perforation of the cecum without the patient being conscious of an increase in the size of the abdomen.

PARTIAL (CHRONIC) OBSTRUCTION OF THE SMALL INTESTINE. A chronic obstruction of the small bowel which, per force, must be a partial obstruction, produces symptoms much less severe than an acute obstruction. The usual symptoms are those of recurrent attacks of crampy pain associated with occasional vomiting which last for a few hours to a day or more. During these periods the patient may notice that his abdomen becomes distended and that he is constipated for a few days. Never do the pains become as severe or the vomiting as frequent or copious as in acute obstructions. Frequently, indiscretions in diet, such as the eating of a particularly large meal or the ingestion of food with a high residue, are noted to bring on these attacks.

PARTIAL (CHRONIC) OBSTRUCTION OF THE COLON. This type of obstruction may exist for a considerable period of time with practically no symptoms, provided the degree of obstruction is not too great. Sooner or later, however, if the degree of obstruction increases, as it usually does in colonic lesions, typical peristaltic cramps occur. If this pain is not

severe, it may be overlooked by the patient for a considerable period of time. Occasionally vomiting may occur but this is not striking. If the obstruction is low in the colon, a decrease in the caliber of the feces is noted. Characteristically, it is said that alternating constipation and diarrhea occurs with this condition. In the author's experience this finding is far from universal and while it frequently occurs too much significance should not be placed on its absence.

STRANGULATION OBSTRUCTION. In addition to those enumerated above, an obstruction of the bowel complicated by strangulation produces other symptoms due to the strangulation. These symptoms are due to a loss of blood into the tissues and lumen of the strangulated segment and peritoneal irritation from blood and bacterial toxins in the peritoneal cavity. Thus, in addition to the symptoms of obstruction are added those of shock and peritonitis. The symptoms of shock vary in severity directly with the length of strangulated bowel and the duration of the obstruction. Signs of peritoneal irritation are localized at first but become general within a few hours. The patient with a typical case, therefore, complains of diffuse abdominal pain and tenderness, prostration and thirst, in addition to his other symptoms.

Physical Examination

GENERAL CONSIDERATION. Examination in the main should be directed to the abdomen. Little is to be learned from the general appearance of the patient. Except in late cases or in the presence of strangulation, the patient with intestinal obstruction does not look particularly ill. If vomiting has been long continued, evidences of dehydration will be noticed. If strangulation is present, the usual signs of shock will be evident.

Patients with bowel obstruction may have a slight elevation of their pulse rate and temperature but not of a sufficient degree to cause alarm except in the presence of strangulation in which the blood loss has been great or a definite peritonitis has developed.

The examination of the heart and lungs is negative unless the abdominal distention is so great that the diaphragm is elevated to such an extent that the cardiac and respiratory functions are compromised.

INSPECTION. The abdomen is usually visibly distended unless the obstruction is high in the small bowel or in the proximal portion of the colon. A considerable distention of several loops of the jejunum may be present without producing a detectable change in the contour of the abdomen. In obese individuals with protuberant abdomens this is particularly true. In such instances, careful attention should be paid to slight degrees of distention in the region of the epigastrium and upper abdomen. Careful inspection of the right lower quadrant of the abdomen may reveal a certain amount of distention which would otherwise

be overlooked in cases of obstruction in the right half of the colon.

The general contours of the abdomen distended with obstructed loops of bowel are not unlike those seen in the presence of ascites. The flanks are broad and bulging and the scaphoid shape of the upper abdomen is replaced by a bulging convexity. Percussion usually serves to make the distinction between these two conditions plain. In cases of obstruction, however, in which the bowel content is largely fluid or in which there has been an unusually large amount of peritoneal fluid, there may be considerable doubt if the differentiation must be made on the basis of the physical examination alone. If the obstruction is chronic in nature or if a chronic obstruction has suddenly become acute and the anterior abdominal wall is thin, individual loops of distended bowel may be seen as corrugations of the abdominal wall and the passage of peristaltic waves along these loops may be noted. The obstruction must be of such a duration as to allow a hypertrophy of the musculature of the intestine to occur before this phenomenon can be perceived.

PALPATION. Palpation of the abdomen of the patient with intestinal obstruction should be primarily directed toward the delineation of any tumor or mass which may be the cause of the obstruction and to determine whether or not there is an inflammatory process within the peritoneal cavity. The latter consideration is extremely important. On the basis of palpation alone the presence of a strangulation obstruction must often be confirmed or excluded. Tenderness, rebound tenderness, and muscle spasm must each be carefully sought for and considered by the physician. In the presence of obstruction the existence of these signs of peritoneal irritation must be taken to indicate the presence of a strangulation unless definite proof exists to the contrary. By the same token, tenderness localized to the vicinity of an incarcerated hernia must be assumed to indicate a strangulation if symptoms of obstruction are also present. It has been intimated that on occasions some degree of peritoneal irritation will be found in cases of simple obstruction if the amount of transudated fluid from the distended loops is great. A certain number of patients may, therefore, be subjected to operation under the mistaken diagnosis of strangulation obstruction. This fact, however, except in very unusual circumstances, should not deter the surgeon from making the diagnosis of strangulation in all cases in which the signs of peritoneal irritation are clear.

The importance of the careful examination of all the usual sites of hernia to detect the existence of incarcerated bowel is easily understood if it is remembered that such lesions account for more cases of obstruction than any other one cause.

AUSCULTATION. No examination can be said to be complete until the surgeon has listened to the abdomen with a stethoscope in a careful and deliberate manner. The existence of borborygmi increased in intensity

and occurring synchronously with crampy pain identifies such pain as due to intestinal colic. This sign is pathognomonic of mechanical intestinal obstruction. Frequently the above fact can be determined within a few seconds; but not rarely, if the interval between peristaltic rushes is prolonged, auscultation must be continued for fifteen or twenty minutes before the examiner can be sure that there is no intestinal colic. It is essential, of course, that the examiner be familiar with the intestinal sounds of the normal abdomen. Judgment must be used in determining whether or not there is an intensification of these sounds. A decrease or even total absence of intestinal noises may be found in cases of *paralytic obstruction*. An increase in the frequency of the *borborygmi* occurs in cases of acute enteritis. In these patients, however, pain is less severe than in those with an obstruction and there is lacking that close association in point of time between the height of the cramp and a loud *borborygmus*. Some observers have attributed a high metallic quality to the typical *borborygmi* of obstruction. This is not always apparent. The quality of the sound appears to depend on many factors, some of which are the strength of the peristaltic rush, the size of the distended loop of bowel and the relative amounts of gas and fluid which it contains.

In partial obstructions these sounds of intestinal colic may be perceived but in a much smaller degree. In colonic obstructions auscultation is a less valuable part of the examination. Typical *borborygmi* may be heard, but in other cases no sounds whatever may be elicited.

RECTAL AND VAGINAL EXAMINATION. Tenderness and the presence of a pelvic mass are the chief points to be determined by vaginal or rectal examination. Not infrequently distended loops of small bowel can be palpated, particularly if their distention is due in large measure to fluid. Quite often the thick-walled, blood-filled coils of a strangulated gut can be detected as a tender mass high in the pelvis. In colonic obstructions the examining finger may be able to determine the exact location and probable character of the obstructing lesion. In cases of intussusception the rectal examination may well be the most important part of the examination. With this lesion the usual signs of peritoneal irritation due to strangulation are absent but the finger may be able to palpate the doughnut-shaped tip of the intussusciens and usually blood can be detected on the gloved finger. The presence of either of these signs increases considerably the likelihood of the diagnosis.

LABORATORY FINDINGS. Simple obstructions *per se* have no distinctive laboratory findings. The urine and blood examinations are normal. If the obstruction has been accompanied by copious and long continued vomiting, the effect of dehydration and chloride loss may clearly manifest itself. The laboratory findings in such cases will show changes due to a concentration of the blood, hypochloremia and extrarenal uremia. The hemoglobin content of the blood is relatively increased. For the

same reason the patient may appear to have a slight leucocytosis. The plasma chlorides are decreased and the carbon dioxide combining power of the blood is increased due to the loss of chlorides in the vomitus. Concomitantly disturbances in the kidney function occur as indicated by an increased blood urea nitrogen and a concentrated urine containing a slight amount of albumen and an occasional hyaline cast.

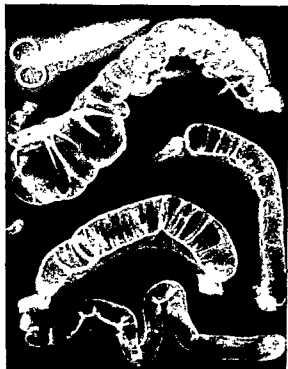


FIG. 185. X-ray film of segments of bowel removed at autopsy and moderately distended with air. 1. Lower ileum. 2. Upper jejunum. 3. Lower jejunum or upper ileum. 4. Cecum and ascending colon. Each part of the bowel has a characteristic appearance when seen on the x-ray film. Knowledge of this fact enables the clinician to estimate the probable position of an intestinal obstruction.

A strangulation obstruction early in its course may not produce any abnormal laboratory findings. If shock ensues, however, the usual signs of hemoconcentration occur. With the development of peritonitis a definite leucocytosis and relative increase in the polymorphonuclears is seen. These latter findings may frequently be of help in making the diagnosis.

GASTRIC CONTENTS AND VOMITUS. Considerable information as to the probable site of the obstructing lesion can be obtained from a gross examination of the vomitus or gastric contents obtained by a duodenal tube. (See section on symptomatology.)

ROENTGEN EXAMINATION. In all suspected cases of intestinal obstruction a roentgenological examination of the abdomen should

be made. The most valuable film is the so-called "scout" film taken with the patient lying in the supine position. Such an exposure delineates the gas containing loops of bowel quite clearly, but magnifies their size by about 25 per cent. Some surgeons have advocated the taking of films with the patient in the upright position and looking for fluid levels which indicate the existence of obstruction. Others have recommended the taking of films with the patient in the prone position. In the author's experience, however, neither of these latter methods give as much or as valuable information as the usual scout (anterior-posterior) exposure.

An unequivocal diagnosis of obstruction should never be made on the basis of x-ray evidence alone. Normally, gas in the small intestine can be definitely visualized on the x-ray film only in infants. Its presence in an adult indicates that there is stasis of the intestinal contents of such a degree that the intimate mixture of fluid and gas has had an opportunity



FIG. 186. Roentgenograms of the abdomen of a patient with a complete obstruction of the small intestine due to adhesions. This film was made at the time of the patient's admission to the hospital. The distention is confined to the stomach and small bowel. The only gas seen in the colon is a few small accumulations in the cecum and ascending colon.

to separate into its respective phases. Only in the broadest sense can this stasis be considered as indicating an obstruction.

Roentgenological findings in cases of mechanical obstruction of both the large and small bowel are fairly characteristic. These findings taken with the other clinical evidence make the diagnosis definite. All clinicians should acquaint themselves with the appearance of distended portions of the various parts of the gastrointestinal tract (Fig. 185). On the basis

of such knowledge the roentgen film can be interpreted accurately and information as to the kind and site of the obstruction with which one has to deal may be obtained.

The presence of loops of distended small bowel and little or no gas in the colon indicates that the obstruction is probably complete and in the



FIG. 187. The same patient as in fig. 186. This film was made forty-eight hours after instituting gastroduodenal aspiration. The aspirating tube has passed into the second portion of the duodenum. The obstruction has been relieved. No gas is seen in the stomach and only a small quantity remains in the small bowel. Considerable gas, however, is now present in the colon.

small bowel (Figs. 186, 187). An approximation as to the probable location of the obstruction in the small bowel can be obtained by noting the mucosal patterns of the distended loops. The restriction of the visible gas to the colon with no loops of distended small bowel seen is the usual picture found in acute obstructions of the colon (Fig. 188). In those instances, however, in which the ileocecal valve is not competent a few distended loops of ileum in addition to a large accumulation of gas in the colon will be found (Fig. 189). Partial obstruction of the small bowel, if at all severe, will exhibit some distention of the proximal bowel

with gas and in addition small scattered accumulations in the colon (Figs. 190, 191, 192). In partial obstruction of the colon there is little or no gas evident in the small bowel and any distention present in the proximal colon is not excessive. The picture in paralytic ileus is variable



FIG. 188. Roentgenogram of the abdomen of a patient with complete obstruction of the sigmoid colon due to a carcinoma. The colon is greatly distended throughout its entire extent. Note the tremendous distension of the cecum as indicated by the arrows. No gas is seen in the small bowel.

(Fig. 193). At times the x-ray picture closely mimics any of the types of obstruction mentioned above. Characteristically, however, stasis occurs in both the colon and small bowel. Gas shadows are seen to be scattered throughout the abdominal cavity. The size of these accumulations depends on the duration and severity of the ileus as well as the quantity of air which the patient has swallowed.

Differential Diagnosis of Intestinal Obstruction

The typical picture of acute small bowel obstruction with colicky pain, vomiting and distention is frequently so characteristic that the diagnosis is not difficult. Other abdominal colics, however, such as biliary,

renal and appendiceal colic must be distinguished. This differentiation is best made with the stethoscope and careful auscultation of the abdomen. The point to be determined is whether or not the intermittent crampy pain is intestinal in origin. If a definite increase in the borborygmi occurs at the height of the pains, it can be correctly assumed



FIG. 189. Roentgenogram of the abdomen of a patient with a complete obstruction of the descending colon due to a carcinoma. The mass of the tumor at the point of obstruction can be seen as indicated by the arrow at the iliac crest. The transverse and ascending colons are moderately distended. The ileocecal valve has allowed the regurgitation of gas into the ileum. As a result a few distended loops of small bowel are seen as indicated by the arrow over the lumbar spine.

that the colic is intestinal in origin. On occasions, an acute gastroenteritis may have to be differentiated. These conditions can at times closely simulate an early acute obstruction. Vomiting associated with a gastroenteritis ceases to be productive as soon as the stomach has been once emptied, provided food and drink are not ingested. Moreover, diarrhea is usually a prominent symptom. The abdominal cramps are usually not as severe as those present in obstruction. In cases in which grave doubt

exists after a careful history and physical examination have been considered roentgen examination of the abdomen usually makes the situation clear.

The diagnosis of chronic small bowel obstruction must be largely made on the basis of a history of previous similar attacks together



FIG. 190. Roentgenograms of the abdomen of a patient with a partial obstruction of the small intestine due to adhesions. (a) This film was made at the time of the patient's admission to the hospital. Accumulations of gas are seen in the stomach, small intestine and colon. Several loops of the small intestine are markedly distended.

with evidence of the existence of an intestinal colic. Other abdominal colics may be satisfactorily excluded by careful auscultation of the abdomen.

The addition of the symptoms and findings of peritoneal irritation to those of an acute obstruction occasion not infrequently considerable difficulty in distinguishing a strangulation obstruction from other in-

flammatory lesions within the abdomen. The conditions which must be considered most seriously are appendicitis, salpingitis, peritonitis, acute pancreatitis, twisted ovarian cyst, ectopic pregnancy and ruptured peptic ulcer. Here again chief reliance should be placed on the presence of intestinal colic and an adequate history. A pelvic examination should



FIG. 191 The same patient as above. Thirty-six hours after instituting gastroduodenal aspiration. The aspirating tube has passed into the first part of the jejunum. The stomach contains no gas and the amount remaining in the small bowel is much less. The colon is now empty.

be most helpful in cases of salpingitis, ovarian cyst and ectopic pregnancy. The x-ray film of the abdomen at times may be the deciding factor in the diagnosis. Certainly in those cases in which a ruptured peptic ulcer is a possibility a film made with the patient in the upright position to show gas under the diaphragm should not be neglected.

Most confusion of mind occurs in differentiating a strangulation obstruction from those cases in which an obstruction supervenes secondary

to an inflammatory process. This condition is probably most often seen when an obstruction occurs during the course of a suppurative peritonitis. Cases will occur from time to time when this distinction is impossible. No effort should be spared to make this differentiation, if possible, because patients with a definite peritonitis are, in the main,



FIG. 192. The same patient as above. Forty-eight hours after instituting gastroduodenal aspiration. The small intestine is now completely empty of gas. Small accumulations are seen in the descending colon. The obstruction has been relieved.

best treated with conservative methods. As will be discussed later, simple obstructions can be adequately controlled and often cured without operation, but strangulations require an immediate operation.

Usually less difficulty exists in distinguishing acute obstructions of the colon from other abdominal conditions. The history is of particular significance. The visualization of distended loops of colon on the x-ray film usually confirms the diagnosis. In late cases in which perforation of the cecum is imminent or has already occurred, an acute suppurative

appendicitis with peritonitis must be excluded. Cases of obstruction of the ascending colon, in which the obstructed segment of the colon is short, are particularly confusing in this respect.



FIG. 193. Roentgenogram of the abdomen of a patient with an inhibitive (paralytic) ileus due to peritonitis secondary to an acute appendicitis. Accumulations of gas are seen in both the small and large bowel. The absence of gas in the right lower quadrant is due to a large appendiceal abscess indicated by the arrow. The other arrows over the lumbar spine indicate accumulations of fluid which separate the distended loops of bowel.

Treatment

GENERAL PRINCIPLES OF TREATMENT. The ultimate aim of all treatment of intestinal obstruction should be the restoration of the continuity of the lumen of the bowel with as little risk to the patient as possible. If modern methods are employed, the time element in the treatment employed has lost most of its importance. Only in cases of strangulation is an immediate operation still imperative.

For practical purposes treatment should include measures to accomplish the following: (1) Decompression of the distention proximal to the

obstruction; (2) restoration of the fluid and electrolyte balance of the body; (3) administration of blood when necessary to overcome any shock present in cases of strangulation; and (4) release of the obstructing mechanism.

RELIEF OF DISTENTION. It has been indicated how distention of the proximal bowel in obstruction produces various secondary effects deleterious to the patient. Its immediate relief, therefore, is all important. There are three methods by which distention can be effectively reduced: (1) Application of a mild, constant, negative pressure to an indwelling

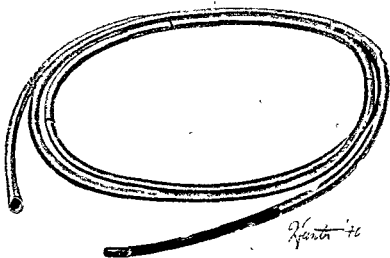


FIG. 194. The Wangenstein modification of the Levin duodenal tube. The distal two and one-half inches of this tube is made of rubber with a specific gravity much greater than that used in the remainder of the tube. Side openings for aspiration extend back approximately nine inches from the tip so that suction can be maintained in both the stomach and duodenum simultaneously.

tube inserted through the nose or mouth into the stomach or duodenum; (2) administration of a high oxygen concentration in the respired air; and (3) enterostomy or colostomy.

APPLICATION OF SUCTION TO INDWELLING TUBES. Wangenstein, in 1931, applied suction to an indwelling duodenal tube in the treatment of certain cases of mechanical obstruction of the small intestine. A modification of the Levin type duodenal tube with perforations extending back from its tip about nine inches and with the distal two inches of the tube weighted by the use of a rubber of high specific gravity was used (Fig. 194). If such a tube is inserted through the nose into the stomach and 70 to 120 cm. of water negative pressure is maintained at its proximal end by a system of bottles as shown in Fig. 195 the stomach is kept empty of all fluid and gas. All vomiting ceases and any distention

already present in the intestine at least ceases to increase and in most instances decreases. Air, as soon as it is swallowed, is removed by the tube. If reverse peristalsis is active and if the pyloric sphincter relaxes

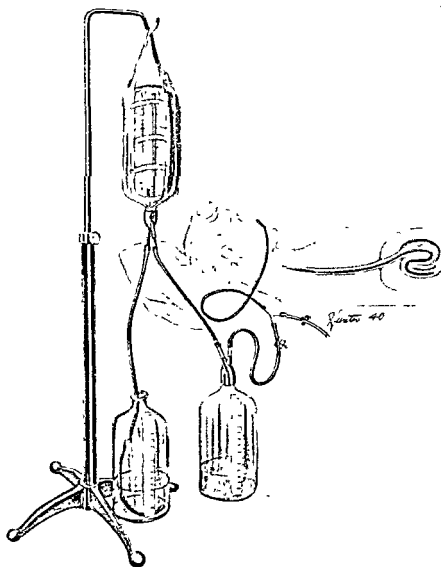


FIG. 195 Apparatus for maintaining a mild constant negative pressure in an indwelling duodenal or intestinal tube. This apparatus can be constructed at little expense from materials available in any hospital. As aspiration progresses, water drains from the upper bottle to the lower bottle which is directly beneath it. The Y tube is used to clear the indwelling tube by the injection of water and thus insure uninterrupted aspiration.

sufficiently to allow free regurgitation into the stomach, small increments of gas and fluid brought up from the lower reaches of the bowel are steadily removed.

The effect of such a tube is increased and more certain in its action if its tip is passed through the pylorus and into the duodenum. Any hindrance to decompression produced by the pyloric sphincter is thus overcome. No certain method exists, however, by which this can be accomplished in every case. The tubes now employed for this purpose are all partially radiopaque so that their position can be accurately determined by the fluoroscope or an x-ray film.

The most effective method of intubating the duodenum is initiated by passing approximately 50 cm. of the tube through the nose and into the stomach. After the stomach has been emptied of its content the patient should be made to lie on his right side. In this position he should drink small quantities of water every few minutes and the tube inserted about one inch every quarter hour until an additional 15 to 20 cm. of tube has been inserted. The slow return of a bile colored fluid when suction is applied usually indicates that the tip of the tube has reached the duodenum. To be sure of this fact, however, an x-ray film of the abdomen should be made. If the tip of the tube on this film is seen to lie to the right of the right border of the vertebral column and pointing downward, it can be assumed that the duodenum has been intubated.

In some cases more than one attempt to intubate the duodenum will have to be made and in rare cases all attempts may result in failure. Some surgeons have adopted the practice of placing the tip of the duodenal tube in the vicinity of the pylorus by manipulation under the fluoroscope. In any event, the actual transit of the tube into the duodenum must be accomplished by the combined effects of gravity and gastric peristalsis.

If the duodenum can be reached, the tube should be inserted four or five inches farther unless a satisfactory decompression is being obtained anyway. The results of this treatment must be closely followed by frequent examinations of the abdomen and by the study of roentgenograms taken every twelve hours. If decompression has not been achieved at least partially within forty-eight hours, an operative decompression should not be delayed further.

Little decompressive effect on a distended colon can be expected from aspiration because of the action of the ileocecal valve. Aspiration will, however, tend to prevent such a distention from becoming larger, since it interrupts the passage of swallowed air through the stomach. If decompression of distention already present is required, aspiration must always be used in conjunction with colostomy.

In 1938, Johnson and his associates advocated the use of a special long double lumen tube (Miller-Abbott tube) with a balloon incorporated in its tip for the decompression of patients with intestinal obstruction (Fig. 196). One of the lumens connects directly with the balloon and the other, which is the larger of the two, is used for aspiration. Such a tube should be inserted through the nose into the stomach

just as the Levin type tube. Likewise, a similar technic should be employed to cause the tube to pass into the duodenum. If this can be achieved, the balloon is inflated with 15 to 20 cm. of air. As more and more of the tube is swallowed peristalsis carries the balloon with the tube trailing behind it farther and farther down the intestine until the point of obstruction is reached. The speed of the tube's passage along the gut seems to depend directly on the force of the peristaltic activity

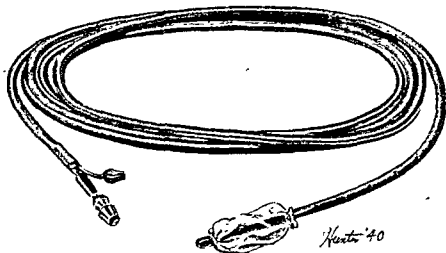


FIG 196. The Miller-Abbott tube for intestinal aspiration. This tube has two lumens. One is used to inflate the small rubber balloon fastened to the distal end while the other is used for aspiration. If the duodenum can be intubated, the balloon is inflated with approximately 15 cc of air or water. Peristalsis will then carry the tube in a few hours well down into the small intestine.

of the bowel. In cases of paralytic ileus intubation may be extremely slow.

The advantages of the Miller-Abbott tube over the Levin type tube as modified by Wangenstein is that decompression of the entire small bowel can be accomplished a great deal more rapidly provided the pyloric sphincter can be passed successfully. Furthermore, if the double lumened tube has been inserted until the tip reaches the point of obstruction and the proximal bowel is decompressed, the patient can ingest and utilize a certain amount of nourishing fluids. Finally, in certain cases, a more accurate diagnosis can be made than would be possible otherwise. If after successful intubation and decompression the point of obstruction is reached, a small quantity of thin barium sulphate suspension injected through the lumen, ordinarily used for aspiration, allows

the roentgenograms to be taken which give valuable information as to the degree, type and exact location of the obstruction.

The chief disadvantage of the Miller-Abbott tube lies in the fact that, although decompression is accomplished as the tube descends through the intestine, distention tends to recur behind it. If swallowed air collects in the stomach and proves troublesome it can be effectively removed only by the insertion of a second (catheter tipped) tube. This is not particularly comfortable for the patient.

The same time limits allowed for decompression by the Wangensteen tube should pertain for the Miller-Abbott tube, namely, forty-eight hours. Likewise, the progress of decompression should be closely followed by frequent examinations of the abdomen and x-ray films.

Patients treated as described above usually require no narcotics. The relief of pain in simple small bowel obstruction is dramatic as soon as the duodenum is decompressed and may occur as soon as the stomach is emptied. A restless patient may appreciate a mild sedative such as sodium pentobarbital, gr. $1\frac{1}{2}$, or sodium phenobarbital, gr. 2, but morphine is not necessary and should not be given. All patients should be encouraged to drink moderate amounts of water and other fluids during the period of aspiration: 2,000 cc. a day is sufficient. Any fluid which will not occlude the aspirating tube may be taken. In this manner the thirst of the patient can be satisfied and the occasional development of parotitis prevented. The injection of a few drops of mineral oil into each nostril twice a day serves to allay, in large measure, the irritation of the nasopharynx due to the long continued presence of the tube. The quantity of fluid aspirated by the tube, as well as the quantity drunk by the patient, must be carefully noted. The difference between these will be the net loss to the patient, and must be taken into account by the surgeon when ordering para-oral fluids.

RESPIRATION OF HIGH OXYGEN CONCENTRATIONS. In 1935, Fine recommended the respiration of a high oxygen concentration for the relief of intestinal distention. To be effective an oxygen concentration of over 80 per cent in the respired air must be employed and its administration must be long continued. Such a concentration cannot be obtained with the usual type of oxygen tent. Fine employed a specially constructed hood but the method was not widely used until Boothby, Lovelace, and Mamoulian developed a face mask by which it was claimed up to 100 per cent oxygen can be cheaply and conveniently given over a prolonged period (Fig. 197).

The principle of this method of treatment depends upon the diffusion of gases from a place of higher concentration to one of lower concentration. It has been previously noted that intestinal gas contains approximately 70 per cent nitrogen. If, therefore, nitrogen be excluded from respired air, that present in the intestine will be gradually absorbed by

the blood and exhaled through the lungs. The other gases, except oxygen found in the intestine, behave in a similar manner. A decrease in the amount of the distention may be noted after a few hours. Fine has also pointed out that such a decrease allows the circulation in the wall of the bowel to improve. Because of the improved circulation it is postulated

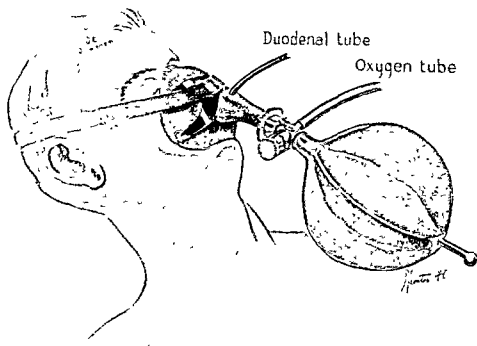


FIG. 197. One type of the Boothby, Lovelace, Mamouliau mask by which high concentrations of oxygen can be respired for relatively long periods of time. This mask has an opening through which an indwelling tube can be passed, so that continuous aspiration of the gastrointestinal tract can be carried out simultaneously. By an adjustment of the valve the concentration of the oxygen respired can be varied. The flow of oxygen should be sufficient so that the attached bag is not entirely empty at the end of inspiration.

that the absorptive power of the gut increases and, therefore, the fluid stagnant in the gut is in part removed and the distention further decreased.

The chief advantage of this method lies in the fact that its action does not depend upon the presence of intestinal peristalsis as does continuous aspiration. It is, therefore, especially indicated in cases of paralytic ileus in which peristalsis is weak or absent.

The disadvantages of the method in the author's opinion outweigh its advantage except in selected cases. Apart from the expense of the oxygen and the discomfort of the patient, if the Boothby mask is employed properly, other factors must be considered. This method does not effectually remove the stagnant fluid which fills to a varying degree the distended coils of intestine in patients with intestinal obstruction. To obviate this difficulty the mask is now constructed with an opening through which an indwelling aspirating tube can be inserted. The possibility of oxygen poisoning, if concentration of oxygen over 80 per cent are respired for thirty-six hours or more, must also be considered. Opinions vary concerning the danger of oxygen poisoning but Fine has recommended that the treatment be interrupted twenty to thirty minutes three times a day to insure against it. This recommendation should be followed.

ENTEROSTOMY AND COLOSTOMY. For those cases of small bowel obstruction in which aspiration has failed to accomplish a decompression in forty-eight hours an enterostomy should be performed. This subject is discussed in a later paragraph. A colostomy placed proximal to the point of obstruction should always be made to obtain a decompression of the distended bowel in cases of obstruction of the colon, unless the obstruction is so close to the cecum that a colostomy cannot be made. In these unusual cases an enterostomy tube can be placed in the terminal ileum and threaded through the ileocecal valve into the cecum. This subject is discussed in a later paragraph.

ADMINISTRATION OF FLUIDS. Fluid administration constitutes a very essential part of the treatment of intestinal obstruction. Administration should be by para-oral routes and should accomplish the following: (1) Supply of a sufficient quantity of water to relieve any dehydration which may be present; (2) supply of an adequate amount of sodium chloride to restore the plasma chloride level to normal; and (3) supply of a daily amount of water and sodium chloride to cover the metabolic needs of the patient until he can safely ingest and absorb liquids.

The types of fluid administered should be normal saline solution and a 5 or 10 per cent glucose solution in distilled water. Either the intravenous or subcutaneous routes may be used. Since, however, relatively large quantities must be given and in many cases to elderly patients whose cardiac reserve is subnormal, the subcutaneous route is favored for routine use in order to obviate the danger of suddenly overloading the right side of the heart. Except in occasional cases in which it may be thought best to return a portion of the aspirated gastric contents, proctoclysis should not be used because of the limited and unpredictable amount of fluid which can be absorbed by the patient in this way.

Coller and Maddock and their associates have shown that the body must lose approximately 6 per cent of its weight in water before clinical

signs of dehydration are evident. To those patients who are thus afflicted an approximation of this amount of fluid should be given slowly over the first twenty-four or thirty-six hours of treatment. The proportion of this large amount of fluid, which should consist of saline solution, will depend upon the degree of hypochloremia which is also present. The remainder of the amount should be given in the form of glucose solutions.

Every patient with intestinal obstruction should have the level of the plasma chlorides determined, if possible, before any plan of treatment is decided upon. Such a determination provides a starting point from which the chloride requirements can be fairly accurately estimated. Coller and Maddock have advised that $\frac{1}{2}$ gm. of sodium chloride per kilogram of body weight in the form of normal saline solution be given for each 100 mg. per cent that the plasma chlorides are below the normal level. Instances occur in which 4,000 to 5,000 cc. of saline solution must be given to satisfy these requirements. A portion, perhaps one-half of such an amount, can be safely given intravenously but the remainder should be injected subcutaneously.

Other factors must be considered in estimating the day-to-day fluid requirements. Newberg has pointed out that a urine output of approximately 1,500 cc. is required to clear the body of the end products of its nitrogenous metabolism if the concentrating powers of the kidney are seriously impaired. It is, therefore, desirable to note the specific gravity of the urine. If this is 1.020 to 1.025, it will be sufficient if the urine output is maintained between 700 to 1,000 cc. The amount of fluid which must be given to produce such an output is dependent on several factors. The most important of these are: (1) fluid lost by evaporation of the perspiration, (2) fluid lost by evaporation through the lungs, and (3) fluid lost by aspiration through an indwelling duodenal and enterostomy tubes. The last of these losses can be accurately measured, but the first two must be estimated. Unless sweating is excessive, 2,500 cc. will usually equal the amount of fluid lost by evaporation from the skin and lungs. The daily volume of fluid required in most cases is somewhat greater than this figure. *The net loss of fluid through the aspirating tube must be considered and also the volume of the urine output desired.* A considerable quantity of water results from the chemical reactions of metabolism, however, so that the actual amount needed is somewhat smaller than might be supposed at first. By experience it has been found that in the average case losing approximately 1,000 cc. of fluid per day through an aspirating tube, a net daily fluid intake of 3,000 to 3,500 cc. is sufficient.

The daily sodium chloride requirements are best determined by frequent estimations of the blood chloride level or the amount of sodium chloride excreted in the urine. A normal level of 560 to 640 mg. per cent of chloride expressed as sodium chloride should be maintained in the

blood plasma. Since chloride is a threshold substance, the body only excretes in the urine that portion for which it has no use. The presence of appreciable amounts of it in the urine, therefore, will not occur unless an adequate supply is available. If the administration of chloride is so regulated that 2 or more gms. of sodium chloride are excreted in the urine passed in twenty-four hours and if that urine is adequate in amount, there is little danger that insufficient quantities of either water or sodium chloride are being given. On the other hand the improper administration of fluids more often results in the giving of too large quantities of salt. Under such circumstances edema develops. Five hundred to 1,000 cc. of normal saline solution in the usual case adequately covers all the chloride requirements unless the amount lost in the fluid aspirated from the stomach is large.

With experience, the surgeon will be able to rely less on laboratory aids and more on clinical evidence, but the proper administration of fluids is a difficult phase of surgery which has not been properly appreciated by the profession as a whole. Many patients have undoubtedly died from the effects of improperly administered saline solution. Edema from this source should be avoided as much as possible. Minimal degrees of this condition usually escape the notice of the physician who does not look for it.

ADMINISTRATION OF BLOOD. No patient with intestinal obstruction should be operated upon in the presence of shock. On the other hand, every patient with intestinal obstruction who is in shock should be operated upon as soon as the shock can be relieved.

The surgeon must learn to distinguish the collapse attending hypochloremia from the shock attending strangulation. Saline solution is almost a specific for the former condition, but the only adequate treatment of shock is the intravenous administration of a sufficient amount of blood, plasma or serum to restore the volume of circulating blood to an amount which will insure a normal blood pressure. No arbitrary amount can be given and called adequate unless the blood pressure and pulse indicate that no more is required.

Not infrequently patients with obstruction due to neoplasms present themselves with marked secondary anemias due to toxemia, chronic bleeding and dietary insufficiencies. These patients are greatly improved as surgical risks by one or more blood transfusions. Such treatment should be given if possible before any operation is performed.

THE NONOPERATIVE TREATMENT OF INTESTINAL OBSTRUCTION. Certain cases of complete simple small bowel obstruction can be treated successfully without operation if an adequate decompression of the proximal bowel can be accomplished by aspiration through an indwelling tube. Wangenstein and Paine, in 1932, reported the first series of cases thus managed. Cases which are amenable to this type of treatment are

usually those in which the obstruction is due to adhesive bands or to a temporary edema secondary to an acute inflammation. Not infrequently both of these processes may be present as in instances occurring during the course of a suppurative appendicitis or salpingitis. Decompression apparently allows a local edema in the walls of the gut at the site of obstruction to subside and the kink in the bowel at this point to straighten out. The method is not successful in every instance and the obstruction may recur; but if it is tried in suitable cases, many patients will be spared the dangers and discomforts of a serious operation.

Decompression may be attempted with the Miller-Abbott tube as previously described. If this is successful, the patient will frequently within twelve to thirty-six hours pass flatus or have a bowel movement. In the absence of either or both of these acts a roentgen film of the abdomen may show the passage of gas into the colon which on previous films appeared empty. Such evidence combined with the nonrecurrence of crampy pain when the aspirating tube is clamped indicate that the lumen of the intestine has reopened. The aspirating tube, however, should not be removed until the patient has demonstrated over a period of hours his ability to ingest liquids without cramps, nausea or vomiting.

Should decompression not be obtained within forty-eight hours, it is best as a rule to resort to operative interference. No attempt should ever be made to treat a strangulating obstruction or a complete obstruction of the colon by aspiration alone. These patients must always be considered as surgical emergencies and operated upon as soon as their condition warrants. Aspiration is carried out in all such cases, but only as a valuable adjunct to the indicated operation. Those cases of simple obstruction in which the nonoperative method is tried but fails should not occasion the surgeons any remorse. If an operation proves necessary in the end, it is one of election rather than one of emergency. The patient's general condition will be much improved by the ample time afforded for adequate preoperative treatment.

OPERATIVE TREATMENT OF INTESTINAL OBSTRUCTION: INDICATIONS. The indications for urgent operative intervention in cases of intestinal obstruction are as follows: (1) The presence of a strangulation obstruction; (2) the development of signs of strangulation in a case of simple mechanical obstruction of the small bowel; (3) the presence of a complete obstruction of the colon; (4) the failure to achieve a decompression of an acute simple mechanical obstruction of the small bowel after forty-eight hours' aspiration through an indwelling tube or the recurrence of such an obstruction after decompression has been accomplished; and (5) the presence of a partial obstruction whose symptoms cannot be relieved by dietary measures or which is caused by a lesion intrinsically dangerous to the life of the patient such as a neoplasm.

PREOPERATIVE TREATMENT. Aspiration through an indwelling tube

should be started on all patients with intestinal obstruction of whatever cause as soon as the diagnosis is made. Other than this, many patients, if seen early in the course of their illness, require no special preoperative treatment. Many patients with acute obstruction of the colon fall in this category and occasionally patients with a strangulated hernia if seen early can be so classified. Most patients, however, are greatly benefited by the preoperative administration of saline and glucose solutions. Patients with strangulations should always be given blood transfusions. The details of these matters have been discussed previously.

If operation is delayed for a day or two, sufficient time will be afforded to correct any deficiency which may be present in vitamin B and C. The importance of an adequate supply of these dietary elements in the surgical patient has been emphasized recently by Holman and others. For patients that require them, 5 mg. of thiamin chloride, 50 mg. of nicotinic acid and 50 mg. of cevitamic acid given twice daily will be sufficient.

The usual preoperative enema can be dispensed with. In case of strangulation it is definitely contraindicated. In all cases, however, care should be taken to see that the patient reaches the operating room with his bladder empty. This is a particularly important consideration in patients to whom large amounts of fluid have been given preoperatively.

PREOPERATIVE MEDICATION. The usual combination of atropine sulphate gr. 1/150 and morphine sulphate gr. 1/6 or 1/4 is quite satisfactory for the average patient. Care must be exercised in the administration of morphine to the aged and very young, due to their increased sensitivity to this drug. In the clinic with which the writer is associated *such patients are routinely given codeine sulphate in appropriate doses* instead of morphine. Likewise the dose of atropine given to young children must be carefully judged. Doses as small as 1/600 gr. are frequently all that is safe and in infants doses of 1/900 gr. are indicated.

The administration of moderate doses of sodium pentobarbital or other barbituric acid derivatives an hour and a half before operation is practiced in many clinics. Such medication appears to decrease the amount of the general anesthetic agent required and is especially helpful if local or spinal anesthesia is to be employed.

CHOICE OF ANESTHETIC. At the present time the surgeon has a wide choice of anesthetic agents from which to select that which is best suited for the patient concerned and the operation to be performed. Many surgeons have favored spinal anesthesia above all others for cases of obstruction. It is usually eminently satisfactory although the author's experience with it has been limited. The continuous aspiration of the gastrointestinal tract which should be routine in the treatment of all cases of obstruction and which should be continued during the period of the operation has removed the fear of regurgitation of gastric contents

and their aspiration into the lungs during an inhalation anesthesia. The author prefers, for the usual case, the inhalation of cyclopropane or ethylene reinforced with ether if necessary.

Although experience is limited, it would appear that in the hands of an experienced anesthetist pentothal given intravenously in combination with curare is satisfactory for abdominal surgery. Obstruction should not contraindicate its use. In critically ill patients, or in those who are poor surgical risks, the amount of general anesthesia required can be greatly decreased and at times even omitted if the local injection of 1 per cent procaine in the abdominal wall is used and if a splanchnic block is done after the abdomen is opened. Some operations such as an enterostomy can be done with only infiltration of the abdominal wall.

INCISION. The surgeon's choice of incision will depend a great deal on the diagnosis of the type of obstruction present. It should be pointed out here that in the presence of an acute obstruction all surgery should primarily be directed toward decompression of the obstructed bowel. The least surgery necessary to accomplish this is the best. Therefore, the two operations most frequently necessary are enterostomy and colostomy. If the obstruction is in the ileum, a low right paramedian incision serves the purpose quite well. The rectus muscle may be split near its medial border or dissected from its sheath and retracted laterally. If the obstruction is in the jejunum, the incision should be placed over the left rectus muscle in the midportion of the abdomen. If a colonic obstruction is present, the best results will be achieved by making the incision over the transverse colon and parallel to it just to the right of the midline. Through this incision the inner fibers of the rectus muscle can be cut transversely and the remainder of the muscle retracted laterally. The exact position of the colon can be determined by taking a film of the abdomen with a coin placed on the umbilicus. For practical purposes, however, if the incision described above is made half way between the umbilicus and the ensiform cartilage, an adequate exposure will be obtained.

If the operation contemplated must be of an exploratory character, a long paramedian incision with splitting of the rectus muscle or retraction of it laterally is best.

ABDOMINAL EXPLORATION. Manipulation of the abdominal contents in the presence of intestinal obstruction must be kept at a minimum. Any surgery performed must be aseptic. Any spillage of intestinal contents is certain to produce death from peritonitis. Why this is true is not definitely known, but two important factors appear: the increase in the number of bacteria in the intestine and the lowered resistance of the peritoneum to infection which occurs as an effect of distention.

The surgeon's curiosity to determine the nature and extent of the obstructing mechanism has in many cases lead to circumstances which

proved fatal to the patient. The friability of the wall of the obstructed distended small bowel is great. It must be handled with great care. If an adhesive band is the cause of the obstruction, the point at which this band impinges on the wall of the bowel is especially liable to be torn by an ill advised exploration.

A certain amount of inspection and manipulation of the abdominal contents is, of course, necessary. The first point to be determined is whether the preoperative diagnosis of large or small bowel obstruction is correct. Having decided which portion of the intestinal tract is obstructed, it must next be determined whether or not a strangulation is present. Usually this question is settled by the presence or absence of a blood tinged peritoneal fluid which may or may not be foul smelling. Occasionally, however, as in cases of intussusception the bowel itself must be examined.

As soon as the questions indicated above have been answered exploration should cease and the indicated operation performed as carefully as possible.

Operative Procedures

RELEASE OF THE OBSTRUCTING MECHANISM. Release of the obstructing mechanism should, as a rule, be attempted only in instances of stran-

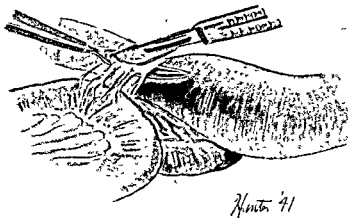


FIG. 198. Method to be employed in cutting an adhesion which has produced a complete obstruction of the small intestine. Such a procedure is dangerous unless the proximal bowel has been previously decompressed by aspiration or enterostomy.

gulation. If that portion of the bowel to which the circulation is obstructed is still viable, release of the obstructing mechanism is all that may be required to cure the patient. If the bowel is nonviable, it will have to be dealt with as explained later. In certain cases of simple obstruction of the small bowel, if the patient is operated upon early, the

surgeon may be justified in seeking out the obstruction and relieving it. Usually this means the division of an adhesive band or constricting ring of peritoneum. The longer the obstruction has been present the more dangerous such a procedure becomes, due to the local necrosis of the bowel at the site of the band or ring. The sudden dilatation of the bowel, when the obstruction is relieved, frequently causes a rupture of its wall at this point and floods the peritoneum with fecal contents.

The technic to be employed should be simple division of the adhesive band with the scissors or scalpel (Fig. 198). If a scalpel is used, a grooved director or Kocher dissector should also be employed to prevent injuring the bowel itself. Particular care must be taken not to cut inadvertently any of the blood vessels of the mesentery. The proper technic for dealing with strangulated hernia is discussed elsewhere in this work.

ENTEROSTOMY (Fig. 199). In the past, enterostomy has been condemned by many as a poor operation, attended by a high mortality and followed frequently by a chronic intestinal fistula. Such a conception arises from errors in technic or from the method by which the operation has been performed. A proper appreciation of the principles inherent in the method first described by Witzel and the use of a catheter no larger than a F.14 or F.16 make enterostomy a safe and extremely useful operation for patients with intestinal obstruction.

The purpose of an enterostomy should be to provide a vent for the escape of gas and fluid from an obstructed small bowel. This may be only a temporary measure which must be followed by another operation to release or remove the obstructing mechanism, but many cases of obstruction due to adhesions will be found to be permanently relieved, or relieved for an indefinite time through the effect of the enterostomy alone. Just as decompression obtained through the agency of an indwelling intestinal tube in certain cases of acute small bowel obstruction relieves the obstruction, so also does decompression obtained through an enterostomy tube. Other uses of enterostomy include the decompression of obstruction of the cecum and ascending colon. In these cases cecostomy may have disadvantages which can be avoided by making an enterostomy immediately proximal to the ileocecal valve and inserting the tube through the valve into the colon. After resections of the bowel an enterostomy made proximal to the site of anastomosis serves as a safety valve for any distention which may occur postoperatively. This affords the best insurance possible against a possible blow-out of the suture line. Frequently, the use of the indwelling tube makes it possible to dispense with an enterostomy for this purpose, however.

A loop of bowel, about eight inches long, a short way above the point of obstruction should be chosen and gently isolated from the remaining coils of intestine with hot, moist laparotomy sponges. This loop should then be emptied of most of its contents by passing two extended fingers

along either side of it and then clamping it lightly at either end with rubber shod intestinal clamps. To remove any remaining gas or fluid, the isolated loop should next be aspirated carefully with a syringe and

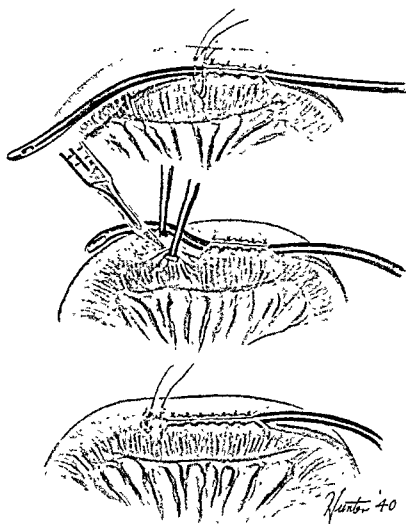


FIG. 190. Technic of enterostomy. The scored catheter is placed parallel with the bowel on the antimesenteric surface and sutured in place. The peritoneal tunnel is begun proximally and continued for about two inches. A small wound through the wall of the bowel enables the catheter to be threaded through into the lumen with no spillage. The peritoneal tunnel is then extended to cover this opening completely. A second row of Halsted mattress sutures of fine silk should be used to insure against leakage.

needle. A dry sponge pressed for a few seconds over the site of the needle hole prevents any contamination from this source. The bowel wall contracts when the loop has been emptied and as it contracts, its wall regains its normal thickness so that it can be sutured safely.

The circumference of the catheter to be used should be scored with

the scalpel about four inches from its tip and a strand of fine catgut tied about it at this point. This suture must not be tied so tightly that it obstructs the lumen of the catheter. The catheter is then clamped at its proximal end and placed parallel with the bowel on its antimesenteric surface. The ends of the suture previously tied about the catheter are then threaded on intestinal needles and stitches taken on either side so that when tied the catheter is inverted within a tunnel of peritoneum. A continuous fine catgut suture is then started about two inches proximal to this first suture and stitches are taken alternately on both sides of the catheter so that when pulled up, it completely covers the catheter in a tunnel of the intestinal wall. This suture is continued to a point about three inches proximal to the tip of the catheter. At this point the bowel should be grasped at either side by Allis forceps and held so that a small stab incision can be made into its lumen. The catheter is then pushed through this opening so that between two and three inches of it extend into the lumen of the bowel. The continuous suture is then extended about two inches farther to prevent any leakage from the stab incision around the catheter. A single stitch of fine silk is next taken beneath the catheter at the proximal end of the tunnel to approximate the bowel tightly about the catheter. Interrupted Halsted mattress sutures of fine silk should then be used to make a second suture line and reinforce the catgut. The proximal end of the catheter can be led out through the incision or a stab wound at one side of the incision in the abdominal wall. If possible, the omentum should be approximated about the catheter as it passes through the abdominal wall.

COLOSTOMY (Fig. 200). Any case of acute obstruction of the colon must be first decompressed through a colostomy before any attempt can safely be made to remove the obstructing lesion itself. Since most obstructions occur in the left half of the colon, a colostomy made in the transverse colon will serve the purpose of decompression just as well as a cecostomy and is to be preferred.

Here again the problem of asepsis must receive the greatest consideration. Even in the best of hands cecostomy is associated with the development of peritonitis in a fair percentage of cases and a moderate mortality. On the other hand, a colostomy can be made in the transverse colon aseptically and with a very low mortality. There remain, however, occasional cases in which the point of obstruction is obviously not suited to a colostomy of this type. Such cases are probably best decompressed by an enterostomy made in the terminal ileum. If this method is employed rather than cecostomy, the surgeon must be sure that the enterostomy tube passes through the ileocecal valve into the cecum or ascending colon.

A transverse colostomy is best made through the incision in the right upper quadrant previously described. When the peritoneal cavity is

opened the distended colon is usually seen or felt immediately below the point of incision. The omentum, if in the way, should be pushed to one side. A short segment of the transverse colon is then herniated into the incision and a glass rod passed beneath it through the mesocolon. Inter-

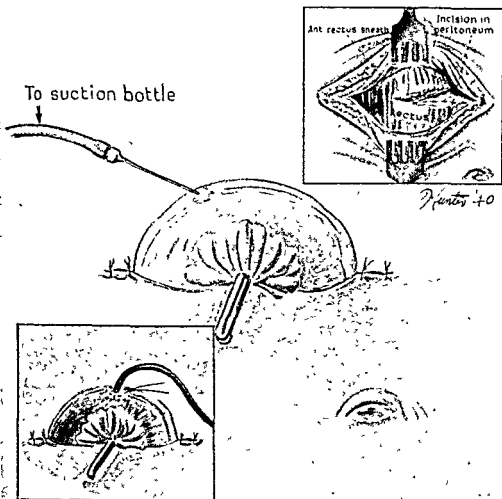


FIG. 200. Technic of making a colostomy in the transverse colon. A portion of the transverse colon has been herniated through a transverse incision just to the right of the mid-line half way between the umbilicus and ensiform process. The medial part of the rectus muscle has been cut transversely. A glass rod placed through the mesocolon holds the bowel in place while interrupted sutures of fine silk are used to suture the peritoneum to the appendices epiploicae and mesocolon. After the operation is completed the distended bowel is decompressed by aspiration through a large needle as seen in the insert.

rupted stitches of fine silk are then placed between the cut edges of the peritoneum and the surrounding mesocolon, omentum and appendices epiploicae. When tied these stitches should effectually prevent any further herniation of the colon or other abdominal viscus. The layers of the abdominal wall require, at the most, only two or three interrupted stitches at each end of the incision to complete the operation.

After vaseline strips have been placed carefully over the incision and all the exteriorized colon, except one small area, a needle connected to a source of negative pressure should be inserted into the lumen of the colon and as much gas and fluid as possible aspirated. This should be repeated every eight hours for the first twenty-four hours. At the end of this time a catheter can be inserted into the colon and fastened with a

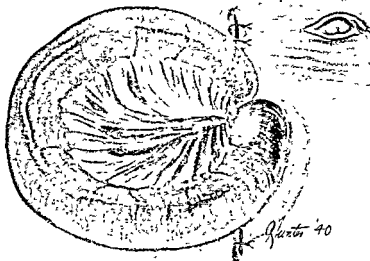


Fig 201. Exteriorization of a loop of small bowel. Such a procedure is proper if the viability of the loop is questionable at the time of operation. If the bowel is gangrenous or becomes so after exteriorization, clamps should be applied to each end a short distance from the skin and the intervening bowel cut away. The clamps can be removed after four to five days when the wound is safely sealed. An enterostomy placed in the bowel just proximal to this point or continuous aspiration through an indwelling tube must be employed to control the obstruction produced while the clamps are in place.

purse-string suture. Irrigations of small quantities of water through this catheter at intervals and continuous aspiration insure a satisfactory control of the distention until the colon can be opened safely with a cautery on the fifth or sixth day after operation.

EXTERIORIZATION (Fig. 201). Exteriorization of a portion of the intestinal tract has been a well established principle of abdominal surgery since Mickulicz and Block independently recommended such a procedure in the treatment of certain carcinomas of the colon. This operation has a definite place in the treatment of strangulation obstruction. It allows the gangrenous bowel to be removed from the peritoneal cavity with as little manipulation and as quickly as possible. It is particularly

suitable for patients critically ill in whom the ability to withstand a prolonged, involved procedure is questionable. Exteriorization, on the other hand, has certain disadvantages. First, and foremost, is the fact that the patient is left at least temporarily with a complete intestinal fistula. For the re-establishment of the continuity of the bowel an additional operation must be performed later.

The potential seriousness of a complete intestinal fistula depends largely on two factors: (1) the level in the bowel at which the fistula is made, and (2) the age of the patient. The higher the fistula is, the greater will be the nutritional problem involved in caring for the patient. Such patients require not only careful supervision of their caloric intake but also meticulous attention to the maintenance of their electrolyte balance. These problems are more difficult of solution in children than in adults. In infants, high intestinal fistulas are extremely serious and are attended with a high mortality despite the utmost of skill and care exercised in their treatment. The surgeon should possess a thorough knowledge of the problems mentioned above and must weigh carefully the advantages and disadvantages pertaining to exteriorization in the individual patient concerned before proceeding with the operation.

If the present enthusiasm for aseptic types of intestinal anastomosis and the development of new technics succeed in decreasing the incidence of peritonitis and the mortality associated with primary resection and anastomosis in the presence of acute obstruction, the indications for exteriorization will be more limited. Despite this, many cases will remain in which exteriorization is the least of several evils when a segment of bowel must be removed to save a life.

Rarely the operator will be unable to decide definitely whether or not a strangulated loop of bowel is viable after the constricting mechanism has been released. In such situations exteriorization offers a middle course between proceeding with a resection which may not be necessary, and closing the abdomen which may contain nonviable gut. If the exteriorized bowel is covered with vaseline dressings, its ultimate survival or death can be awaited with equanimity. If time brings recovery, the abdominal incision can be reopened and the bowel returned to the peritoneal cavity with very little risk to the patient.

The technic of exteriorization is not difficult. After the gangrenous loop of intestine has been freed from any surrounding adhesions, it, together with two or three inches of normal bowel at either end, is lifted to the exterior of the abdomen. Any serosanguineous fluid in the peritoneal cavity should be aspirated. If circumstances permit, the mesentery of the normal bowel at the ends of the gangrenous loop should be approximated with interrupted stitches. On one side the normal bowel and on the other the mesentery of the exteriorized bowel should be fastened to the adjacent peritoneum to prevent any further evisceration.

The ends of the incision should then be closed so that only the index finger can be passed into the peritoneal cavity with facility. The mesentery of the bowel itself should be left under as little tension as possible. Vaseline strips should be used to cover the exteriorized bowel and incision.

If the exteriorized bowel is frankly gangrenous, it had best be excised

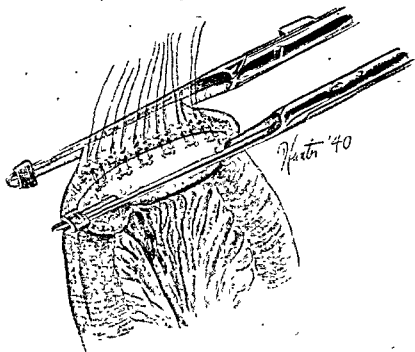


FIG. 202. Aseptic side-to-side anastomosis of the small intestine after a resection has been performed. The ends of the bowel have been closed. The intestinal clamps have been applied to the side of the bowel, and the outer row of Halsted mattress sutures on the posterior surface have been applied.

immediately. For this purpose, heavy intestinal clamps applied to the viable portions of the exteriorized gut adjoining each end of the gangrenous loop and left in place for as long a period as possible afford the best assurance against a wound infection or peritonitis. Under such circumstances provision must be made for decompression of the obstructed proximal bowel. Aspiration through an indwelling tube may prove sufficient. However, the insertion of a catheter as an enterostomy, by the technic described elsewhere in this chapter, immediately proximal to the proximal clamp is a safer procedure. Another alternative is the insertion of a large tube into the lumen of the proximal bowel and the ligation of the bowel about it. By this arrangement the intestinal contents can be

collected and returned to the distal bowel for assimilation and absorption.

RESECTION AND PRIMARY ANASTOMOSIS. Because of the individual's poor resistance to peritonitis from intestinal contamination when obstruction is present, resection of the bowel and primary anastomosis, except when performed aseptically, are fraught with danger and are at-

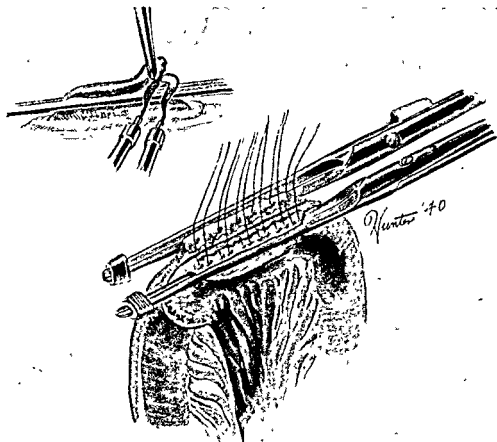


FIG. 203. Same as previous figure. The inner row of Cushing mattress sutures on the posterior surface has been inserted. The cautery is used to remove a small portion of the bowel wall projecting beyond the edge of the clamps.

tended by an extremely high mortality. Some writers have placed the mortality of such procedures as high as 85 to 90 per cent. Preliminary decompression either by an indwelling tube, an enterostomy or a colostomy, as the case indicates, however, changes the picture considerably. A decompressed bowel can be resected and an anastomosis performed with only a moderate risk to the patient.

Resection of bowel in the presence of an acute obstruction of either the small intestine or colon, should be considered only in cases of strangulation and should be performed only when the surgeon has decided

that the great risks attending this procedure are less than those involved in exteriorization. Specifically, resection and anastomosis will be indicated only in those rare cases of strangulation involving the jejunum in adults, and the ileum or jejunum in young children and infants.

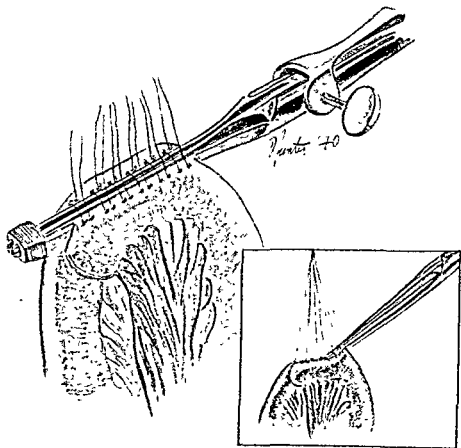


FIG. 204 Same as previous figure. The clamps have been rotated inward and fastened together with ferrule and clamp. The inner row of Cushing mattress sutures on the anterior surface has been placed but not tied. As clamps are withdrawn, tension is maintained on these sutures. They are then tied.

After preliminary decompression has been obtained, the indications for resection in dealing with a persisting obstructing mechanism embrace a much wider variety of lesions. Typically, these include carcinomas, certain benign tumors, benign stenoses from various causes and dense multiple adhesions.

The technic employed should avoid opening the bowel and should be carried out aseptically. Wangenstein has described such a method, which clinical experience has proved to be quite satisfactory. This

method is adaptable to any type of anastomosis anywhere in the gastro-intestinal tract. Figures 202 to 209 show the steps in this method for end-to-end, end-to-side and side-to-side anastomoses. For a detailed description of the method the reader should consult the original article noted in the bibliography.

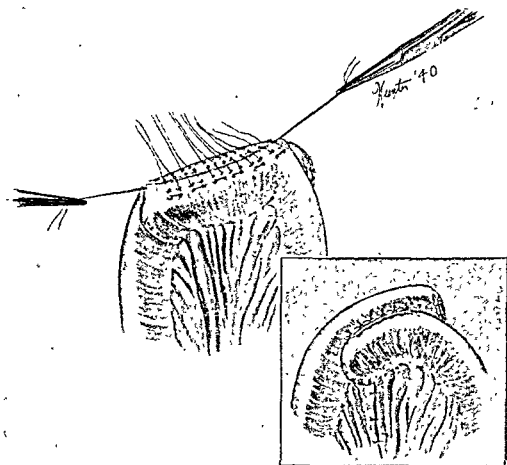


FIG. 205. Same as previous figure. The anastomosis is finished with the outer row of Halsted mattress sutures on the anterior surface. The thumb and forefinger should be herniated through the site of the anastomosis to open the bowel where it has been crushed by the clamps. Reinforcing sutures should be placed at each end of the anastomosis to prevent tension on the suture lines.

The principles of this method are as follows: Two modified Martzloff-Burget clamps with narrow blades and a ferrule to hold the opposed blades of the clamps in line are used. In addition a locking device and double ferrule are employed to approximate the two clamps while the anterior layers of sutures are being inserted. Two layers of sutures are used, those on the posterior surface being placed first with the intestinal clamps rotated outward (Figs. 202, 203), and those on the anterior surface being placed after the clamps have been rotated inward and locked

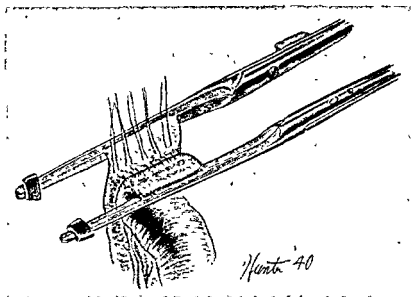


FIG. 206. End-to-end anastomosis of the bowel after segmental resection. To insure the vitality of the antimesenteric portion of the bowel, it should be cut slightly oblique. Halsted mattress sutures are inserted about 1 cm from the intestinal clamps which are turned outward. These form the outer layer of sutures on the posterior surface.

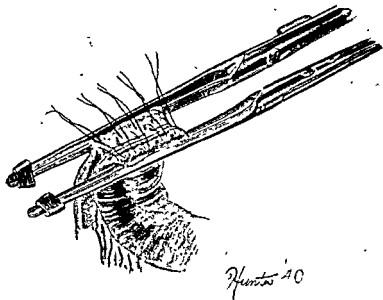


FIG. 207. Same as previous figure. The Halsted mattress sutures have been tied. A line of Cushing mattress stitches are used to form the inner layer of sutures on the posterior surface.

with ferrule and clamp (Fig. 204). Interrupted sutures of fine surgical silk may be used throughout the procedure, although the inner layer can be made with a continuous suture of fine chromic catgut on an atraumatic needle if the operator desires. After the inner line of stitches on the anterior surface has been placed, but not tied, the clamps are loosened and gentle traction made on the sutures as the clamps are with-

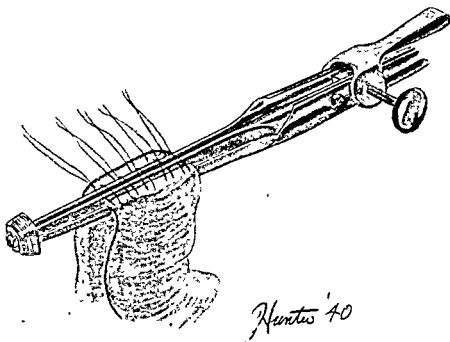


FIG. 208. Same as previous figure. The posterior sutures have been placed and tied. The intestinal clamps have been rotated inward and clamped together. The inner layer of sutures composed of Cushing mattress stitches has been placed.

drawn (Fig. 204, ins.). This maneuver closes the anastomosis and eliminates any chance of spillage as the sutures are tied, one by one, while traction is maintained on the others. The outer reinforcing layer is then inserted and tied in the usual manner (Fig. 205). At the completion of the operation the fingers are worked back and forth at the site of the anastomosis to open the ends of the bowel which have been crushed by the clamps.

Usually, an oblique end-to-end anastomosis will be preferable to another type in those cases in which a segment of bowel is to be removed (Figs. 206, 207, 208, 209). The bowel should be cut with the cautery immediately adjacent to the clamps to avoid any chance of contamination. If the above described technic is employed in performing a side-to-side anastomosis, the clamps must be applied to the side of the bowel and

must enclose within their blades some of the mucosa as well as the muscularis of the intestinal wall. The projection of the bowel beyond the clamp is then removed with the cautery and the sutures applied as described above.

ENTEROANASTOMOSIS. When obstruction is produced by a lesion such

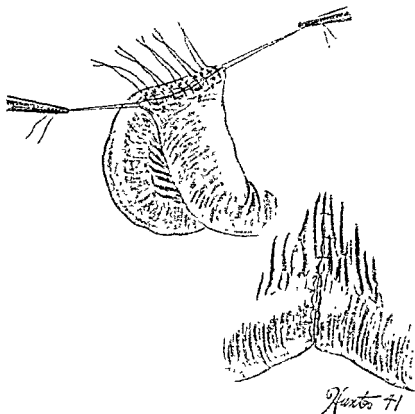


FIG. 209. Same as previous figure. The stitches forming the inner layer on the anterior surface have been tied after the clamps were withdrawn. Traction sutures at each end of the anastomosis are held taut while the anastomosis is completed with a row of Halsted mattress sutures. The operation is completed by sewing the cut edges of the mesentery together.

as a malignant tumor which cannot be removed, the continuity of the intestinal tract may be restored by a side to side anastomosis between the proximal and distal segments of bowel (Fig. 210). Likewise, such a procedure will be in order occasionally in cases in which the obstruction is due to adhesions which are too numerous or dense to be severed and in which a resection of this portion of bowel is contraindicated.

In these cases the technic described in the preceding paragraphs should be used.

POSTOPERATIVE TREATMENT: CARE OF THE WOUND. The care of the abdominal wound should be similar to that of any laparotomy wound. If there is no drainage and convalescence is uneventful, the dressings applied in the operating room at the completion of the operation need not be disturbed for nine or ten days. At this time they should be re-

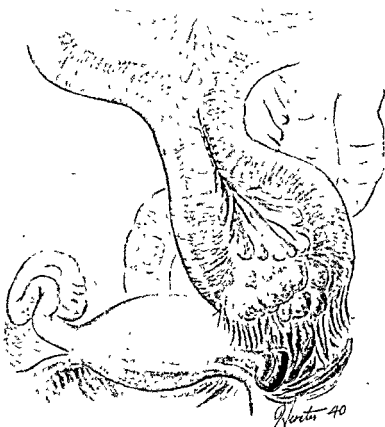


FIG. 210. A carcinoma of the ovary which has involved a loop of ileum by direct extension and produced a partial obstruction. A side-to-side anastomosis has been performed to short circuit the intestinal tract and relieve the obstructive symptoms. If such a tumor can be removed, the ileum should be resected and an anastomosis made to re-establish the continuity of the intestine.

moved and the skin sutures removed. If primary healing has occurred, further dressings will be required for only an additional twenty-four to forty-eight hours.

Any enterostomy tube should be kept fastened securely to the skin of the abdomen with adhesive tape as well as to the dressings applied over the incision. Especial care must be exercised not to remove the tube inadvertently when the abdominal dressings are changed.

If bowel has been exteriorized as in a colostomy or after the exteriori-

zation of bowel of questionable viability, it should be kept covered with strips of vaseline gauze. In addition, similar strips should be applied closely over the points at which bowel and skin come in contact to prevent contamination and infection of the abdominal wall and peritoneal cavity. In addition, gauze and absorbent pads should be applied over these vaseline dressings. This more or less bulky dressing must be removed from time to time, as indicated, for examination. For those cases in which an entire loop of bowel has been exteriorized, a further protection in the form of a cardboard box with the walls cut to approximate the contours of the abdomen can be employed.

Rarely after a colostomy, but always after a fistula of the small bowel has been made, provision must be taken to protect the surrounding skin from irritation due to the fecal discharge. There is no agent or method which will control this situation ideally. Various salves and preparations have been recommended. The author employs 5 to 10 per cent tannic acid in vaseline as an ointment applied frequently and liberally with fairly satisfactory results.

MEDICATIONS. As after any abdominal operation patients with intestinal obstruction, who are operated upon, require sedatives and narcotics to relieve their pain in the immediate postoperative period. Morphine sulfate or codein hydrochloride in appropriate doses according to the age, size and general condition of the patient are effective and generally quite satisfactory. In order to prevent addiction, the use of such narcotics should be closely supervised after the first forty-eight hours. As soon as possible, milder sedatives should be substituted to control any restlessness which may remain after the acute pain of the operation has subsided. Sodium phenobarbital in 2 gr. doses is quite effective if hypodermic administration is necessary; otherwise, sodium pentobarbital, gr. 1½, sodium amytal, gr. 2, as well as other barbituric acid derivatives can be used.

The use of prostigmine, mecholyl and other drugs alleged to increase peristalsis and minimize gas pains is not necessary if gastroduodenal aspiration is maintained routinely for three or four days after operation. The author does not use such drugs at all and would advise against their use except in very exceptional cases.

It is well to give one or two ounces of mineral oil daily as soon as gastroduodenal aspiration is discontinued and the patient begins to eat. Such a régime frequently eliminates the necessity of giving enemas and increases the patient's comfort until normal bowel habits are resumed.

As previously indicated the development of peritonitis following operations performed on the bowel in the presence of acute intestinal obstruction is a most important factor contributing to the notorious high mortality of this condition. The use of the sulphonamide compounds and penicillin may be of help in decreasing the incidence of infection and

thereby reducing the mortality. Results in a few cases personally witnessed by the author led him to believe that there is a place for these drugs in the treatment of certain cases of intestinal obstruction.

ASPIRATION OF THE GASTROINTESTINAL TRACT BY SUCTION APPLIED TO AN INDWELLING TUBE. All patients with an obstruction for which an operation has been necessary should have suction applied to an indwelling tube continued for two to four days after the operation. No arbitrary limit can be set at which such treatment can be discontinued. The purposes of this form of treatment and the principles of its employment, previously discussed, must be kept in mind. This type of treatment gives the great protection to anastomotic suture lines and controls the distention of paralytic ileus better than any drug.

ADMINISTRATION OF FLUIDS. The principles of fluid administration have been covered in some detail. Postoperatively, fluids must be supplied by para-oral routes until intestinal aspiration is discontinued and an adequate oral intake is possible. The loss of chlorides through aspiration as well as that which may occur from an intestinal fistula must be compensated for by the administration of normal saline solution. The remainder of the fluid requirement should be supplied in the form of 5 or 10 per cent glucose solution in distilled water.

Special Remarks on the Various Anatomic Types of Intestinal Obstruction

1. **ATRESIA OF THE INTESTINAL TRACT.** This subject is dealt with elsewhere in this work.

2. **IMPERFORATE ANUS.** This subject is dealt with elsewhere in this work.

3. **TUMORS AND STRICTURES OF THE BOWEL WALL.** Both benign and malignant tumors developing in the wall of the bowel may produce obstruction. All clinicians are well aware of the frequency of colonic obstruction due to a constricting carcinoma. In the colon such cases are far more frequent than obstruction produced by a benign tumor. On the other hand, in the small intestine the incidence of carcinoma is relatively rare. Many more cases of obstruction of this portion of the bowel are due to benign tumors than to carcinomas. Of the nonmalignant strictures of the bowel which produce obstruction those due to tuberculosis are the most frequent. Other causes of these lesions are syphilis and ulceration due to the various types of enteritis. In the distal half of the colon an appreciable number of strictures are due to diverticulitis. Among other causes may be included infarction of a small segment of bowel which survives by revascularization from the omentum, fibrosis at the point of constriction after reduction of an incarcerated hernia, fibrosis of the bowel wall following x-ray radiation of the abdomen and regional enteritis.

Inasmuch as the obstruction produced by these lesions develops slowly, the symptoms of a partial obstruction may be recognized and treatment begun before obstruction becomes complete. On the other hand, many patients wait until the severe symptoms of a complete occlusion supervene before seeking medical advice. Not infrequently, even the physician may overlook the existence of a partial obstruction if the symptoms are mild and intermittent in their occurrence. Even if suspected, negative evidence furnished by the x-ray, after a barium meal or enema, may cause such a diagnosis to be excluded from consideration unless it is borne in mind that benign strictures may be extremely difficult to demonstrate in this manner.

If the diagnosis can be established before the obstruction becomes complete, the patient should be treated in a conservative manner until a remission of symptoms occurs. At that time an operation of election should be performed and the obstructing lesion dealt with directly. The choice of operative procedures in such cases will lie between a side-to-side anastomosis of the gut, so that the fecal stream is short-circuited around the obstructing lesion, and a segmental resection of the lesion followed by some type of anastomosis to re-establish the continuity of the intestinal lumen. The latter procedure is to be preferred unless excision of the lesion is impossible.

In instances of acute obstruction of the colon or small intestine due to either a tumor or stricture, decompression of the distended proximal bowel must first be accomplished before the obstructing lesion itself can be safely attacked. For lesions in the small bowel decompression by means of intestinal aspiration should first be attempted. If this is successful, the obstruction can be attacked directly and dealt with as indicated above for partial obstructions. If decompression by aspiration is not successful, a preliminary enterostomy should be performed and the obstructing mechanism dealt with later at a second operation within ten days to two weeks. If the obstruction is in the colon, decompression through a colostomy should always precede an operation designed to re-establish the continuity of the intestinal tract.

4. **OBTURATION OBSTRUCTION:** (a) *Obstruction Due to Gallstones* (Fig. 211). Following the formation of a fistula between the duodenum and the gallbladder, gallstones of large size may gain entrance to the bowel and produce a simple mechanical obstruction. The lower ileum is most frequently the site of such obstructions since that portion of the intestinal tract has the smallest diameter. Most cases of this nature are seen in middle or old age. Women are affected about three times as frequently as men.

Characteristically, the patient presents a long history of biliary tract disease with a recent severe attack. Jaundice may or may not have been present. For the first day or two, the symptoms may be of an intermit-

tent obstructive character as the stone attempts to pass through the small bowel. If impaction occurs, the symptoms of a complete obstruction quickly develop. Ulceration may occur at the site of impaction and in late unrelieved cases this may progress to perforation and be the cause of peritonitis.

The correct diagnosis can be suspected at times from the history and



FIG. 211. Roentgenogram of the abdomen of a patient with a partial obstruction of the small intestine due to gallstones. A fistula has formed between the gallbladder and duodenum. Gas can be seen in the bile duct. There is some distention of the small bowel, but considerable gas is also seen in the colon. The gallstones can be seen in the mid lower portion of the abdomen.

confirmation can be obtained if the gallstone can be visualized on a roentgenogram of the abdomen. Not infrequently, however, even the most careful study of such films fails to reveal the stone. Suggestive evidence of a gallstone obstruction is offered by the visualization of air in the biliary ducts. This finding, of course, merely indicates the presence of a fistula between the duct system and the intestinal tract, but is fre-

quently seen on films of the abdomen of patients with gallstone obstruction.

† Treatment should include an enterotomy and removal of the stone. A right rectus incision is satisfactory. An attempt should be made to move the stone proximally in the bowel before it is opened so that the enterotomy incision can be placed in a portion of the intestine which has not had its viability compromised by pressure due to a prolonged impaction. In some cases this is impossible and the incision must be made at the original site of impaction. That segment of the bowel containing the stone should then be isolated between two rubber covered intestinal clamps. A line of interrupted Halstead mattress sutures should then be placed transverse to the long axis of the bowel and should extend about half of the way around the circumference. The ends of each suture should be tagged separately with a small hemostat and the middle portion of the sutures themselves retracted off the surface of the bowel wall with skin hooks while the enterotomy incision is made in the center. As soon as the stone is removed the sutures are released from the skin hooks and traction made on all the sutures at the same time by elevating the attached hemostats. This closes the opening in the bowel immediately and diminishes as much as possible the danger of fecal contamination. The separate stitches may then be tied one at a time and the line of closure reinforced with another layer of the same kind of sutures.

The mortality of gallstone obstruction is high, being about 50 per cent in most reported series. The technic described above, first used by Wangenstein, should improve the outlook of this condition if the diagnosis can be made reasonably early.

(b) *Foreign Bodies and Food.* A variety of foreign bodies and various types of ingested material have been reported on occasions as being the cause of simple mechanical obstruction. Many of these cases have occurred among inmates of mental institutions. In years past, Murphy buttons used for intestinal anastomoses have failed to pass and produced obstructions. Accumulations of fruit seeds and grain husks are the more important causes of obstruction due to ingested foods.

In general, these cases are rare. Either a partial or complete obstruction may occur. Treatment should include the operative removal of the obstructing material through an enterotomy. A technic similar to that described for the removal of an obstructing gallstone is advisable.

(c) *Enteroliths.* Intestinal obstruction due to an enterolith is rare. A few cases are reported, however, in which alkaline salts of minerals have been deposited around the nucleus of a foreign body and produced a stone large enough to occlude the lumen of the intestine. Such lesions occur in the colon and seem to be secondary to chronic fecal stasis.

Symptoms of either a complete or partial obstruction may be present. Treatment should include the removal of the enterolith by a technic

similar to that described for the removal of an obstructing gallstone.

(d) *Worms*. This rare condition is seen primarily in children, but an occasional case may be met with in adults. The *ascaris lumbricoides* is the parasite most frequently responsible. Obstruction occurs in the distal portion of the ileum and may be either complete or partial in character.

Treatment should include removal of the obstructing mass of worms through an enterotomy as previously described or by resection of the involved portion of intestine.

5. **MEGACOLON**. The cause of this condition is not fully understood. It may make its first appearance in adult life but usually has its inception in childhood. The bowel is enormously dilated and the wall hypertrophied. Obstruction occurs secondary to chronic fecal stasis and the gradual accumulation of inspissated fecal masses. A careful history is important if a correct diagnosis is to be made. The striking features found on physical examination are a protuberant abdomen and large palpable fecal masses.

Treatment of this condition after obstruction has developed should consist of a preliminary colostomy to decompress the colon and provide a means by which the fecal content can be removed. Later a bilateral lumbar sympathectomy may be performed. If this does not relieve the condition, a resection of the colon should be done.

6. **EXTRINSIC COMPRESSION OF THE BOWEL**. Obstruction of the bowel due to extrinsic pressure occurs usually at those points at which the bowel is firmly fixed to surrounding structures. Tumors of the pelvic organs and large tumors of the mesentery are the most frequent causes. The obstruction is usually partial in character.

Treatment should include the operative removal of the obstructive mass, if possible. If such a procedure is not feasible, a short-circuiting anastomosis can be constructed. Some tumors of the mesentery which may produce obstruction respond well to radiation therapy.

7. **ADHESIONS AND FIBROUS BANDS**. Intraperitoneal adhesions and fibrous bands share with hernia the distinction of being the most frequent cause of mechanical obstruction of the small bowel. The relative frequency of these lesions in representative series of cases has been indicated in Table II. Adhesions and bands may be congenital in origin, the result of peritonitis or the result of trauma to the peritoneum at the time of a previous laparotomy. The type of obstruction produced may be simple or strangulating in character, partial or complete in degree. Obstructions occurring in the immediate postoperative period are practically all due to this cause.

Because of the variation in the type of obstruction produced the treatment must be much more individualized than for other obstructive conditions. Many cases in this category, however, in which the obstruction

is simple in character, can be successfully treated by continuous aspiration through an indwelling tube without operative intervention. Of course, the obstructive mechanism remains unaltered by such a procedure, but the obstruction to the passage of the bowel contents is overcome. No assurance can be given, however, that similar attacks will not occur subsequently. As previously indicated, unless such patients are operated upon very early, search for and liberation of the obstructed loop of bowel is dangerous. In cases without strangulation, decompression by intestinal aspiration should be attempted first. Failing in this, an enterostomy should be performed. A secondary operation can be carried out later if need be and the adhesions cut or the involved segment of bowel resected. If strangulation is present, the involved bowel must be either exteriorized or resected.

8. EXTERNAL HERNIA. This subject is dealt with elsewhere in this work.

9. INTERNAL HERNIA. This subject is dealt with elsewhere in this work.

10. VOLVULUS. There is a great geographical variation in the frequency of volvulus as a cause of intestinal obstruction. Many more cases are seen in eastern Europe than elsewhere. Most series show that it is the cause of about 10 per cent of all obstructions. The small intestine, the sigmoid flexure of the colon and the cecum are the usual sites of this lesion. A potential strangulating type of obstruction results.

If this condition is recognized and operated upon early before definite infarction occurs, untwisting the involved loop of bowel may be all that is required. It should be borne in mind, however, that recurrence of a volvulus of the sigmoid colon is not infrequent. More radical therapeutic procedures will be necessitated when the involved bowel is found not to be viable. These include exteriorization and excision with subsequent anastomosis and primary resection and anastomosis.

11. INTUSSUSCEPTION. A discussion of this condition as it occurs in children will be found elsewhere in this work.

Seventy-five per cent of all cases of intussusception occur in children under two years of age. In adults the usual causes are tumors developing in the wall of the bowel. These tumors include polyps, leiomyomas, lipomas and fibromas. The small bowel is more frequently the site of the primary lesion than is the colon.

The presence of mucus and blood in the feces associated with the usual symptoms and signs of a simple obstruction constitute an almost unique symptom complex. Despite the fact that this lesion produces an infarction of a portion of the involved bowel, the usual signs of strangulation are absent due to the fact that the circulation of the outer layer of bowel is not compromised. For this reason the serosanguineous exudation into the peritoneal cavity, characteristic of strangulations, fails to occur. The

palpation of a sausage-shaped tumor within the abdominal cavity lends confirmation to a suspected diagnosis. If the apex of the intussusception has progressed so that it enters the colon, roentgenological examination of the abdomen with a barium enema usually demonstrates the lesion beyond question.

All patients with intussusception should be operated upon as soon as the diagnosis is made. In many instances the intussusception can be manually reduced. Decision as to the viability of the bowel must be made at the time of operation. If the bowel is viable, the excision of the primary lesion should be postponed for ten days or two weeks, in order that the risks attending resection or exteriorization of the bowel in the presence of an acute obstruction may be avoided. It must be remembered, however, that the intussusception may recur during this period of waiting. Those patients in whom the bowel is found to be nonviable must be subjected to resection or exteriorization.

12. **ERRORS IN THE DEVELOPMENT OF THE BOWEL.** Most cases of obstruction due to errors in the development of the bowel occur in childhood and are discussed elsewhere in this work. Cases of obstruction due to a Meckel's diverticulum, however, must be placed in this classification and many of these cases occur in adult life. The actual cause of the obstruction is usually a fibrous band running from the apex of the diverticulum to the anterior abdominal wall or to a loop of small bowel. Either a simple or strangulating type of obstruction may be produced. Such cases should be treated according to the principles discussed for the treatment of obstructions due to adhesions and fibrous bands.

13. **INHIBITION ILEUS (PARALYTIC ILEUS).** This condition is fundamentally due to an overactivity of the sympathetic nerves supplying the bowel. It constitutes the most frequent cause of intestinal obstruction considered in its broadest sense. The obstruction is entirely functional in character and is due to the absence of normal peristaltic movements. The causes of inhibition ileus are many and include the following:

Peritonitis	Infectious fevers
Intra-abdominal operations	Injuries of the central nervous system
External trauma of the abdomen	Injuries to the spine
Acute abdominal colics	Injuries of the chest
Torsions of abdominal viscera	Thoracic operations
Retroperitoneal lesions	

The patient complains of no abdominal pain. There is no intestinal colic. Auscultation of the abdomen reveals relatively few borborygmi. Abdominal distention is always present, but the degree of the distention and the extent of the bowel involved in it is subject to wide variation.

Frequently, the occurrence of an inhibition ileus can be prognosticated. In such cases treatment should be begun before distention occurs

rather than afterwards. The best treatment is to employ constant suction to an indwelling duodenal or intestinal tube. This will remove all swallowed air as it passes into the stomach and intestine as well as a considerable proportion of that gas which is already present in the bowel. Cases in which the distention has become well established before suction is started occasionally present difficult therapeutic problems. Decompression from above is made more difficult by the absence of effective peristaltic movements. In such cases the addition of the administration of high oxygen concentrations, as described by Fine, may be of benefit. Spinal anesthesia to remove temporarily the effect of the sympathetic impulses on the musculature of the bowel wall has been recommended but the effect generally is only transitory. The intravenous injection of a hypertonic saline solution may also give temporary relief. A proper quantity for a patient weighing about 70 kilograms would be 75 cc. of a 15 per cent solution.

The judicious use of constant suction applied to an indwelling tube has largely eliminated the necessity of performing enterostomies for this condition. Nevertheless, rare cases may be seen in which this method of decompression is justified. The surgeon should remember, however, that the obstruction due to this condition is both functional and temporary. For the control of distention of the colon in this condition, reliance must not be placed on suction, but rather on the periodic use of a rectal tube, enemas and the respiration of a high oxygen concentration.

14. **SPASTIC ILEUS (DYNAMIC ILEUS).** Rare cases of obstruction are encountered usually in neurotic women in whom a persisting spasm of a segment of the bowel is great enough to occlude its lumen. It is thought that in such instances some dysfunction of the intrinsic nerves of the bowel is at fault.

The clinical features of the condition are those of a simple mechanical obstruction. Intestinal colic is always present. The diagnosis cannot be made with certainty except by operation.

If the obstruction is in the small bowel, continuous suction alone may control the condition until the spasm relaxes; otherwise, an enterostomy should be performed. If the point of obstruction is in the colon, a colostomy should be made.

15. **MESENTERIC THROMBOSIS AND EMBOLISM.** Occlusion of the mesenteric arteries by thrombosis occurs less frequently than occlusion by emboli. The superior mesenteric artery is more frequently involved than the inferior. The sources of practically all emboli affecting these vessels are vegetations within the left half of the heart and thrombi within the left auricle. Thrombosis of the mesenteric veins generally occurs in association with infectious processes in the area drained by the affected vessel.

Most cases of obstruction from these causes occur in adults with some

type of heart disease. The onset is typically acute and accompanied by severe abdominal pain, vomiting and diarrhea. Although at times definite colicky pain is present, this symptom is much less prominent than in certain other types of obstruction. The exudation of an irritating serosanguineous fluid from the infarcted segment of bowel occurs rapidly and usually produces shock and peritoneal irritation. With the passage of time if the vessel is large and completely occluded, gangrene and perforation of the affected segment of the intestine must occur.

A definite diagnosis is not usually made prior to operation. The differentiation of this condition from an acute pancreatitis or ruptured viscus is difficult.

Treatment must include a laparotomy preceded by adequate transfusions to relieve any shock which may be present. The involved bowel must be either excised or exteriorized. In rare instances, short segments of bowel of questionable viability may be encountered. Such a segment should be exteriorized but left intact. If it remains viable after a few days, it can be returned to the abdominal cavity. The decision between resection and exteriorization must be made by the surgeon and will be based on the circumstances present in each case.

MORTALITY. Up to 1930 the reported general mortality in all large series of cases of intestinal obstruction, not including paralytic ileus, was about 50 per cent. The few series reported since then, however, have shown a notable decrease as indicated below:

	No. of Cases	Dates	Per Cent
Van Buren and Smith	130	1932-1935	28.4
Schlicke, Borgen and Dixon	133	1938-1939	21.8
Kennedy and Hanson	139	1932-1938	24.4
Wangensteen, Rea, Smith and Schwyzer	190	1932-1939	14.7

Deaver and Moynihan each believed that a mortality of 10 per cent was attainable in this condition. Such a result seems a long way off at present, but there is much hope that the progress which began to bear fruit in the thirties will be continued.

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Chapter XIV

Urgent Intestinal Surgery in Infants

By JULIUS L. SPIVACK

There are several affections of the gastrointestinal tract in infants which are incompatible with life, unless they are remedied by surgical means. Some urgently require correction two or three days after birth, if success is to be realized, while others permit a delay of at least a few days.

Congenital Atresia and Stenosis of the Small Intestine and Colon

These two conditions have in common not only the similarity of their embryologic origin but also a similar pathologic basis and symptomatology. The latter is only more pronounced in atresia than stenosis, but their therapeutic management is essentially identical. The prognosis, however, is better in stenosis than in cases of atresia.

EMBRYOLOGIC AND PATHOLOGIC CONSIDERATIONS. Prior to the fifth week of intra-uterine life the intestinal lumen of the fetus is lined with epithelium. Starting from the fifth week the epithelium begins to proliferate until it fills the entire lumen of the intestine, and then begins to shrink so that by the tenth week the bowel presents a solid cord. From the eleventh week vacuoles appear in the center of the cord; they become larger, coalesce with each other until a lumen is reestablished. If the formation of vacuoles at some portion does not occur, local atresia will take place. If vacuoles at some place develop only imperfectly, a diaphragm will be present, producing constriction of the lumen of the particular loop, the degree of which depends on the size of the opening in the diaphragm.

Atresia is found in two forms. In the most frequent one, the intestine ends in a blind sac, so that the distended proximal loop is not connected with the rest of the tract (Fig. 212). In the less frequent form, the lumen is totally obstructed by a diaphragm; however, there is a continuity of the intestinal wall proximally and distally to the diaphragm. There may be more than one atresia loop (Fig. 212 (I)). The bowel, proximal to the atresia, is greatly distended, reaching as much as 3 to 4 cm. in diameter, while the distal loop is collapsed reaching only 5 to 6 mm. in diameter.

SYMPTOMATOLOGY. The symptoms vary in intensity depending on whether they are due to atresia or constriction, being milder in the latter. They usually appear on the first day of life, the earliest symptom being vomitus. The character of its contents depends on the site of the

obstruction. If it is in the duodenum above the ampulla of Vater it consists only of milk and does not contain bile. As most of the duodenal obstructions are below the ampulla, the vomitus contains bile and pancreatic juice of a greenish color. If the seat of obstruction is in the lower ileum or in the colon, the vomited material has a fecal odor and appearance.

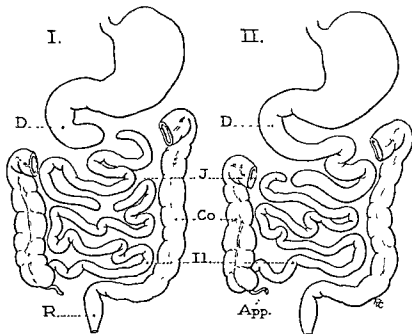


FIG. 212. Different types of atresia of the intestine. I. Isolated blind loops causing multiple obstructions II. Single obstruction. D—duodenum; J—jejunum; Il—ileum; Co—colon; R—rectum; App—appendix.

Abdominal distention may or may not be present, depending on the seat of the obstruction. If the obstruction is in the duodenum, the distention is limited to the epigastric region. But if the infant vomits violently the epigastrium may not be distended. If the atresia is in the jejunum and particularly in the ileum the abdominal distention is generalized. The distention may appear immediately after birth, if the infant swallowed enough of amniotic fluid, or between 24 to 48 hours after birth depending on the seat of obstruction, the amount of ingested milk, and the degree of the vomitus.

Fever is usually moderate ranging at 100–101° F. A temperature above 102° F. suggests the possibility of peritonitis.

Stool. The meconium in atresia differs from the normal. Ordinarily, infants swallow some amniotic fluid in which, among other substances, are desquamated epithelial cells from the skin of the fetus. These cornified cells are also present in normal meconium but absent in atresia. Basing on this consideration, Farber offered a test which enables one

to differentiate these cornified cells in the meconium and their presence or absence is a deciding factor in differentiating between atresia, when these cells are absent, and partial constriction when they are present.

Roentgenologic examination, as a rule, is not necessary as the clinical diagnosis is not difficult. It may aid occasionally in determining the site of the lesion. A flat x-ray plate shows the distended loops. Barium should not be given.

The diagnosis is based on the vomitus, starting the first or second day, abdominal distention and the characteristic absence of cornified cells in the meconium.

TREATMENT. The patient should be operated as soon as possible. If the operation is done 72 hours after birth, peritonitis or even perforation is already present. *The mortality, even under most favorable conditions, is still high.* The prognosis, however, is considerably better in case of stricture than of atresia. However, with a prompt diagnosis and preoperative management, the outlook for saving the infant's life is now considerably better than it was in previous years.

Preoperative measures consist of giving the infant 10 per cent glucose intravenously before the operation in the amount of 10 cc. per pound of body weight or of 15 cc. of normal salt solution per pound of body weight, given subcutaneously. This should be followed by a blood transfusion, 10 cc. per each pound of body weight, to be given after the operation.

Gastric lavage to decompress the stomach should be carried out immediately before operation, using a tube No. 8 French scale.

The infant's arm and legs should be wrapped to conserve body heat.

A hot water bottle should be placed under the back.

OPERATIVE TECHNIC. Anesthesia may be general or local. We prefer ether by the open drop method. A very small amount is needed in order to have the abdomen relaxed.

A four-inch right paramedian abdominal incision should be made with the upper two inches above and the lower two inches below the umbilicus. This incision will afford access to any part of the bowel. After the abdomen is opened there will be, as a rule, escape of free fluid. If, however, it is cloudy or malodorous, one should suspect a perforation and search for it. The seat of the atresia or stricture is found by recalling that loops proximal to stricture are distended and those distal collapsed. The distended loops float and have a tendency to protrude outside the abdominal cavity. Their walls are thin and may be ruptured by rough manipulations.

The management depends on whether there is gangrene, perforation, multiple atresia and also on the site of atresia. If the proximal loop is gangrenous or perforated, it has to be resected and the continuity established by a side-to-side anastomosis with closure of the ends (Figs. 213,

214). End-to-end anastomosis should never be attempted because there is great inequality of the lumina of the proximal and distal ends and also because the wall of the proximal loop is thin and leakage is liable to occur particularly by the effort of the proximal loop to propel intestinal con-

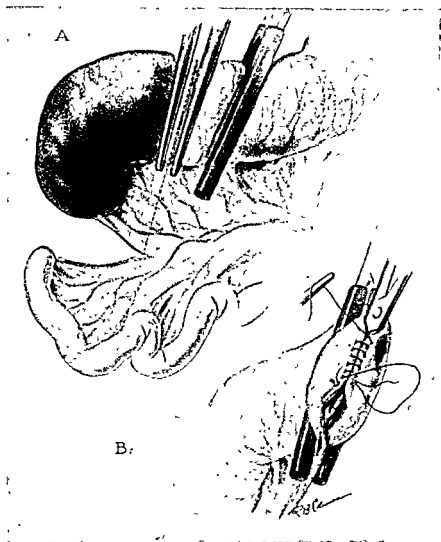


FIG. 213. Operative management when proximal loop is gangrenous and resection necessary. A. Proximal loop clamped—dotted line shows site of incision for removal of gangrenous portion of bowel. B. Cut end of proximal loop closed blindly over clamp by continuous over and over suture.

tents through the narrowed stoma. If there is no gangrene or perforation the atresic part does not necessarily need to be resected in the same sitting. For the time being it will suffice to unite the loop just proximal to the atresia with the loop distal to the obstruction by a lateral anastomosis in two layers; the first, as a sero-serous, done with Pagenstecher

linen and the second as a through-and-through union, with chromic cat-gut 0000 (Fig. 215).

As there is great disparity between the diameter of the proximal and

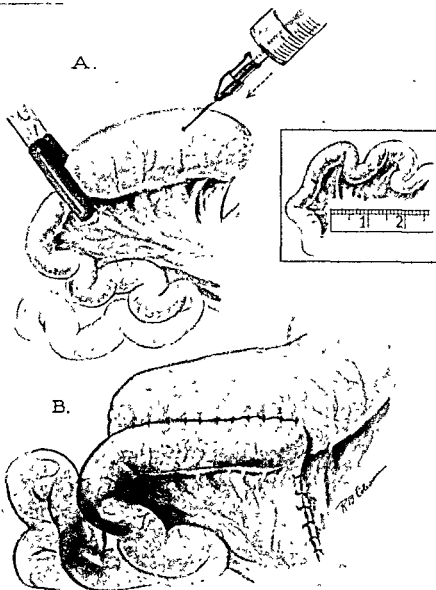


FIG. 214. Operative treatment of intestinal atresia. A. Injection of air into collapsed distal loop. B. Side-to-side anastomosis between the proximal and distal loops. Insert. Actual size of a collapsed loop before injection with air.

that of the distal loops, it is advisable to aspirate the air and liquid contents of the proximal loop and inject air into the distal loop before starting the anastomosis (Fig. 214). One should also not overlook the possibility of the presence of atresias at several places; therefore, the entire length of the bowel should be examined and an anastomosis made be-

tween the loop immediately proximal to the most proximal atresic loop and the loop immediately distal to the most distal atresic loop (Fig. 215). The intermediate loop of the bowel may either be resected at once or at some future time.

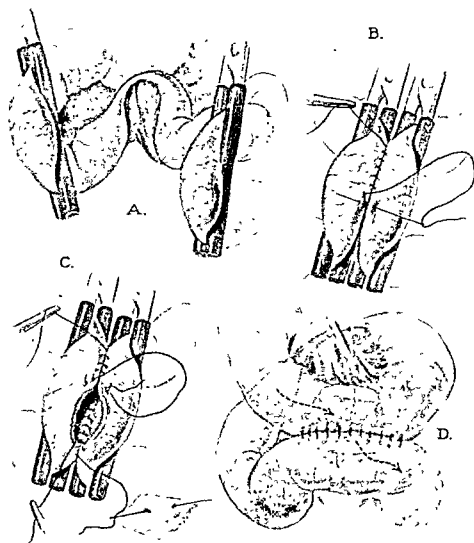


FIG. 215. Intestinal atresia. Isoperistaltic lateral anastomosis for cases of intestinal constriction. Operative treatment: A. Intestinal clamps on distal and proximal sides of constriction. B. Loops of bowel in clamps placed side by side in isoperistaltic direction. First layer of sutures (seromuscular) being placed. C. Anterior through-and-through sutures. D. Completed anastomosis showing sidetracking of constricted area. Arrows indicate new pathway for passage of intestinal contents.

If there is constriction of bowel, the best procedure is to sidetrack the constricted portion and perform a lateral anastomosis, taking one loop

immediately above and the other immediately below the site of stricture. The suturing is done in two layers—the first seroserous with fine Pagenstecher linen thread and the other as a through-and-through suture with 0000 chromic catgut. As in the case of atresia, to reduce the disparity between the lumina of the loops, one may inject air with a syringe into the collapsed bowel and aspirate the contents from the distended proximal loop with a needle before starting the anastomosis.

In some cases the surgeon may be tempted to perform an emergency enterostomy in order to decompress the proximal loops with the hope of reconstructing the continuity of the intestinal tract at some future time. Statistical study shows that this procedure is accompanied by a mortality of nearly one hundred per cent, particularly if obstruction is in the jejunum. The reason for this mortality is extreme and rapid dehydration which takes place in spite of energetic parenteral administration of glucose, saline solution, plasma or blood. However, if the operator has not much experience in intestinal suturing and if the obstruction is in the lower ileum, he may confine himself to a low ileostomy as the first stage, to be followed, not later than 48 hours, by a reestablishment of the continuity of the intestinal tract.

If the obstruction is in the duodenum it is important, before selecting parts for anastomosis, to decide whether the atresia is above or below the ampulla of Vater.

If it is above the ampulla—gastrojejunostomy is the operation of choice as the bile and pancreatic juice flow distally without hindrance. If, however, the obstruction is below the ampulla, duodenojejunostomy should be done, uniting a portion of the duodenum immediately above the site of obstruction with a high jejunal loop. In this variation, if gastrojejunostomy were done, bile and pancreatic juice would find their way to the rest of the gastrointestinal tract by passing first through the pylorus into the stomach and from there through the gastrojejunal stoma into the bowel.

POSTOPERATIVE MANAGEMENT. The patient should receive fluids parenterally two or three times daily. Gastric lavage should be made repeatedly as long as vomiting persists. The colon should daily be dilated with saline enemas. Blood or plasma transfusion should be given immediately after the operation and thereafter as often as may prove necessary to maintain a normal blood count and plasma protein level.

THE MORTALITY in cases of atresia is as high as 85 per cent and in strictures reaches about 60 per cent. The reasons for this high mortality are clear: the condition is comparatively rare and very often is recognized only late. The little patient often is moribund before operation. Often the wall of the distended loop is very thin; the distal loop has a lumen of the size of a small lead pencil; therefore, even if an anastomosis is performed, the thick meconium has difficulty in passing through a very

narrow intestinal lumen. The only hope for a lowered mortality lies in early diagnosis, proper preoperative and postoperative management and gentle operative technic.

Malrotation of the Intestines (Figs. 216, 217)

Malrotation of the intestine implies either insufficient rotation of the bowel in embryonic life, or arrest of its descent or failure to develop sufficient peritoneal attachments so as to keep the bowels fairly immobile. These conditions may produce intestinal obstruction.

In some instances there may be no symptoms pointing to this affection, but in most cases intestinal obstruction arises sooner or later, necessitating surgical intervention. To W. E. Ladd belongs the credit for showing how simply this condition can be remedied, if recognized. He reduced the mortality rate to a fair prognostic outlook. A clear idea of the mechanism producing this condition is conveyed by the following:

EMBRYOLOGIC CONSIDERATIONS. The gastro-intestinal tract of the embryo is subdivided into three parts: the foregut, from which develops the stomach, the midgut, from which develops the duodenum, jejunum, ileum, cecum and the appendix, colon ascendens and the right half of the transverse colon, and the hindgut, from which develops the remainder of the large bowel. Between the 6th and 10th weeks of embryonic life the alimentary tube grows faster than the peritoneal cavity, as a result of which a portion of the bowel protrudes outside the abdominal cavity and becomes situated at the base of the umbilical cord. The portion of the bowel extending from the duodenum to the vitelline duct (Meckel's diverticulum) lies in front of the superior mesenteric artery, while the remaining portion of the bowel lies behind that artery.

From the 10th week on, the abdominal cavity begins to grow faster than the bowels, with the result that the latter recede from the outside into the peritoneal cavity and rotate in counter clock direction, so that the portion of the bowel which was in front of the superior mesenteric artery lies to the right, and the remainder of the bowel to the left of the superior mesenteric artery.

With the growth of the embryo and the progress of the rotation, the cecum and colon ascendens reach the epigastrium, lying under the stomach and still later in the right upper quadrant under the liver. Later, the cecum descends and occupies the upper portion of the right iliac fossa, and the ascending colon reaches the lumbar region. After rotation and descent are completed, the cecum and colon attain their normal peritoneal attachment and the small bowel its attachment to the posterior abdominal wall along a line running from the duodenojejunal junction to the ileocecal junction.

If there is an arrest in rotation of the gut, the cecum may remain in any intermediate position. If the cecum remains under the stomach or

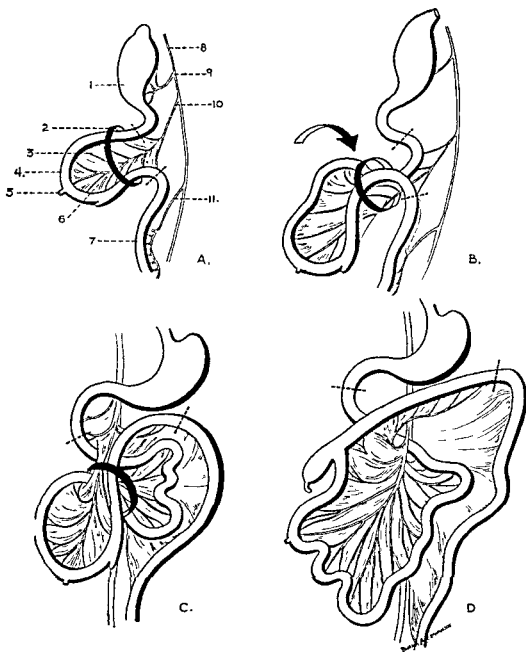


FIG. 216. Rotation of the intestine in embryo. 1. Foregut. 2. Umbilical orifice. 3. Prearterial mesentery. 4. Midgut. 5. Site of vitelline duct. 6. Postarterial mesentery. 7. Hindgut. 8. Aorta. 9. Coeliac artery. 10. Superior mesenteric artery. 11. Inferior mesenteric artery. A. Primitive alimentary tract at 5th week. B. Condition of alimentary tract at about 8th week. The first stage of rotation is being completed. C. Condition of alimentary tract at 10th week. The second stage is in progress. D. Condition of alimentary tract at about the 11th week. The second stage of rotation is completed.

under the liver, it may compress by its weight a portion of the duodenum lying behind it; more often, however, the peritoneal attachment running from the cecum to the postero-lateral abdominal wall in front of the duodenum may compress the latter and produce a high intestinal ob-

struction. Even if the cecum descends and reaches its normal position it may in some cases remain extremely mobile due to lack of development of the peritoneal attachment; in other cases not only does the cecum remain mobile, but the small bowel is not attached by a mesen-

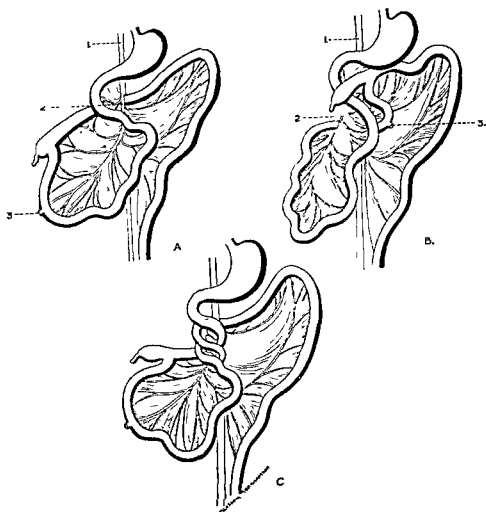


FIG 217. Malrotation of the intestine 1. Aorta 2. Superior mesenteric artery. 3. Vitelline duct. A. Reversed rotation of midgut loop. B. Malrotation of midgut loop. Reversed rotation of the prearterial segment to 90° in a clockwise direction. C. Volvulus of the midgut loop. The twist in a clockwise direction of 150° .

teric root to the posterior abdominal wall, the connection being only through a small band lying immediately below the superior mesenteric artery, thus leaving the small bowel very movable and easily subject to volvulus.

All these conditions, namely, compression of the duodenum by the

cecum, volvulus of the jejunum, ileum or cecum may produce intestinal obstruction.

PATHOLOGY. The pathology depends on the character of the embryologic deviation from the normal. If the cecum does not descend, it either compresses the second or the third portion of the duodenum or a peritoneal band, stretching between the cecum and the posterior-lateral portion of the abdominal wall, compresses the duodenum. If the pathologic condition is due to a limited attachment of the small bowel to the posterior abdominal wall, there is volvulus, usually in a clockwise direction, in which the bowel may turn from 180 degrees to four complete turns. A characteristic feature in this case is that after abdomen is opened no cecum or colon ascendens are seen, because it is covered by loops of small bowel, and in one part it seems that an intestinal loop has herniated through a hole in the mesentery of another loop.

One should bear in mind that non-descent of the cecum and volvulus of the ileum may coexist, as well as normally placed but a movable cecum may coexist with a movable and only partially attached mesentery of the small intestine.

SYMPTOMATOLOGY. The symptoms vary depending on the site and degree of obstruction. Vomitus is the most prominent symptom. It usually contains bile of a greenish color, as the obstruction in most cases is below the ampulla of Vater.

The feces are scant in partial obstruction and absent in complete obstruction.

Abdominal distention is marked in the epigastric region in case of duodenal compression, and in the umbilical or in the entire abdomen in volvulus of the small intestine. In duodenal obstruction the degree of distention may be slight, if the vomiting is propulsive.

Dehydration rapidly develops due to the vomitus of gastric, biliary and pancreatic juices.

Fever is moderate, due to dehydration. However, if peritonitis or intestinal infarcts occur, the fever may reach as high as 105° F.

Hemoconcentration is usually present and is of moderate degree due to dehydration. If, however, marked leukocytosis is present, one should think of infarction of the bowel.

X-ray. A plain film will show a gas-filled stomach and duodenum and only few air bubbles below the duodenum not only in case of compression of the duodenum but also in case of volvulus. However, in the latter case the small bowels may also be distended.

The diagnosis is based on the clinical picture of intestinal obstruction and on roentgenologic findings. If the condition occurs within the first few days of life it should be differentiated from atresia or constriction of the bowel.

TREATMENT. In all cases of malrotation with symptoms of intestinal obstruction the treatment is surgical. As in all other conditions in children, proper preoperative care, minimum manipulations during operation and supportive postoperative treatment will considerably reduce the mortality.

Preoperative management consists of decompression of the stomach by inserting through the nostril into the stomach a rubber catheter No. 8 French, which is done immediately preceding the operation and by giving 10 per cent glucose intravenously or of saline solution either intravenously or subcutaneously.

OPERATIVE TECHNIC. Ether anesthesia by the open drop method. This is preferable to local analgesia, as complete relaxation is important.

Incision. The abdomen is entered through a right paramedian incision, extending from the xyphoid process to one inch below the umbilicus. After the abdomen is open, in case of intestinal obstruction, some fluid escapes. If it is cloudy or of foul odor, perforation may be present and should be looked for.

The appearance of the contents in the abdominal cavity will give a clue as to the character of the pathologic process, and determine the technic:

a. If the cecum lies in the epigastric region and the duodenum is distended, the cause of obstruction is compression of the duodenum either by the cecum or by a band extending from the cecum to the lateral abdominal wall. Any bands are cut laterally from the cecum together with the posterior parietal peritoneum. This will relieve compression and the cecum will be transposed medially. Even if bands are not seen and obstruction is due to compression by the cecum itself, the parietal peritoneum lateral to the cecum should be incised and the gut transferred mediad and be left in this position.

b. If the cecum and colon are not visible and only loops of distended small bowels with bluish color are seen, then one may expect to find volvulus of the small intestines; one usually will find as if one loop of bowel herniated through the mesenteric opening of another loop of bowel. In such a case Ladd suggested and carried out many times the following technic: The entire small bowel is delivered outside the abdominal cavity. As the volvulus occurs in a clockwise direction, the reduction should be made in the opposite direction. After that the bluish color disappears. It should be emphasized that no attempts at reducing the volvulus should be made until the entire length of the bowel is brought outside the abdominal cavity, which is a simple procedure, as the mesentery is attached to the posterior abdominal wall only by a small band. If only part of the bowel is delivered outside and an attempt is made to disentangle the bowel, confusion easily results. When the volvulus is disengaged, one should always ascertain before closing

the abdomen the position of the cecum and colon. If the cecum and colon lie in the right upper quadrant (and this often is the case), they must be freed from their position, by incising the lateral parietal peritoneum and then transposed to the left side of the abdomen, where they remain. Many deaths were due to the fact that the surgeon relieved the volvulus and failed to relieve the concomitant duodenal obstruction produced by the cecum.

c. If after the abdomen is opened a mobile cecum, lacking a posterior abdominal attachment, is found, one has to ascertain whether there is a normal oblique attachment of small bowel to the posterior abdominal wall. If this attachment is present, the cecum should be fixed posteriorly by a few sutures. If, however, the attachment of the small bowel occurs only through a small bundle below the superior mesenteric artery, the parietal peritoneum lateral to the ascending colon and hepatic flexure should be slit and the entire right colon (cecum, colon ascendens and right half of the transverse colon) transferred to the left part of the abdomen.

MORTALITY RATE. While the mortality rate stated by many authors is very high, Ladd by using the above described technic was able to reduce his mortality to 25 per cent.

Meckel's Diverticulum (Figs. 218-223)

Meckel's diverticulum is found in about two per cent of all autopsies. As the number of cases coming to a surgeon due to complications is comparatively small it is evident that complications arise very infrequently, but if they do it is usually in the first two years of infancy. However, it may become a source of surgical complications at any age for which reason, if encountered during laparotomy, the diverticulum should be removed.

PATHOLOGY. Meckel's diverticulum is a pouch from one half to three inches long, arising in most of the cases from the antimesenteric border of the ileum at a distance of 18 to 36 inches from the ileocecal junction. When seen by the surgeon its mucosa is identical with that of the neighboring ileal mucosa in about 30 per cent. In the rest, it has in addition to the normal ileal mucosa also some gastric mucosa (in about 50 per cent), colon mucosa or pancreatic tissue. The pathologic conditions arising in the abdomen are due to several causes. Perforation of the diverticulum or hemorrhage through rupture of its vessels may occur after peptic ulceration of the aberrant gastric mucosa. In other cases the apex of the diverticulum may adhere to the abdominal wall or to some intra-abdominal viscus, forming a ring through which another loop of bowel may be caught and produce intestinal obstruction (Figs. 218, 219). In another group the diverticulum may become a starting point for intussusception (Fig. 220) and in still other cases a volvulus of

the pouch may take place (Fig. 221). In rare cases Meckel's pouch may retain its early embryologic characteristics, communicating with the yolk sac; in other words, there is present a fistula connecting the umbilicus with the ileum through the diverticulum (Fig. 218, D).

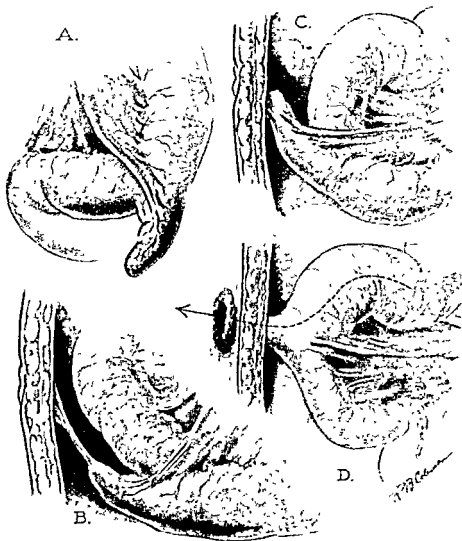


FIG. 218. Meckel's diverticulum. A. Diverticulum laying free on the antimesenteric border. B. Diverticulum attached to the umbilicus by a fibrous band. C. Diverticulum attached directly to the umbilicus. D. Diverticulum forming a fistula at the umbilicus.

THE SYMPTOMS vary according to the character of the pathologic process. Therefore, the surgeon may be confronted by symptoms of intestinal perforation, hemorrhage, intestinal obstruction or peritonitis. Persistent fecal discharge will be present at the umbilicus in case of a non-obiterated vitelline duct.

X-ray examination is of no particular value, as in many cases diverticula are not demonstrable even after a barium meal.

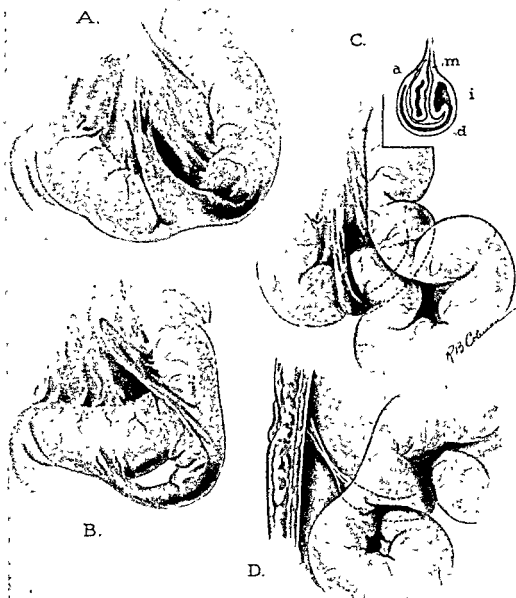


FIG. 219. Meckel's diverticulum. A. Diverticulum attached to the mesentery. B. Distal end of the diverticulum attached to the bowel. C. Diverticulum passing behind a loop of ileum and its mesentery returns back over ileum and attaches to ileal mesentery. D. Diverticulum occluding an intestinal loop between its origin and fibrous attachment to the umbilicus. a. adhesion. d. diverticulum. i. intestine. m. mesentery.

TREATMENT. In a non-complicated case, when the general condition of the patient is good excision of the diverticulum and closure of the opening in the bowel suffices (Fig. 222). In order not to constrict the lumen of the bowel, it may be closed in a direction perpendicular to the

long axis of the ileum (Fig. 223). The closure is done in two layers—the first being a through-and-through suture with chromic catgut 0000, and the second a sero-serosa layer with Pagenstecher linen thread. In intussusception, the diverticulum should be first reduced, further action de-

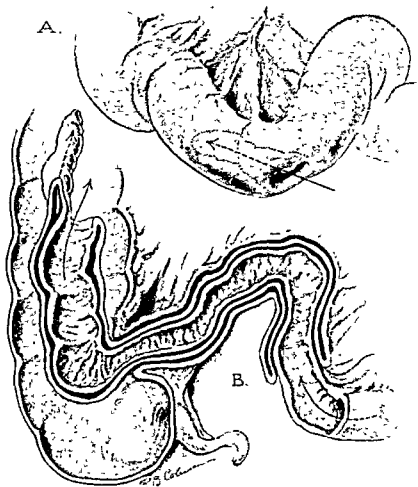


FIG 220. Meckel's diverticulum. A. Diverticulum inverted into lumen of ileum—starting point for intussusception. B. Semi-diagrammatic cross section (frontal) showing more advanced intussusception which began with inversion of Meckel's diverticulum.

pending on the state of the wall of the diverticulum and of the adjoining ileum. If it is not damaged, diverticulectomy should be carried out as just described. The same technic is employed if the wall of the diverticulum is damaged. If, however, there is damage to the adjoining intestinal wall, the damaged loop together with the diverticulum should be extra-

peritonized, brought outside the skin and removed in a few days, depending on the condition of the patient. The continuity of the gastrointestinal tract is reestablished as early as possible. This will be discussed more in detail in the section on intussusception.

THE MORTALITY rate depends on the character of the pathologic pro-

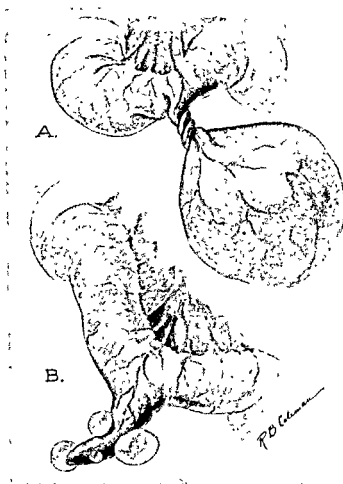


FIG. 221. Meckel's diverticulum. A. Volvulus of the diverticulum. B. Diverticulum lying free with several false diverticula.

ess, when operation was performed, and the condition of the patient before operation. It averages about 30 to 35 per cent.

Intussusception

This is one of the most frequent surgical emergencies in infants. Its symptoms and course are quite characteristic and if early recognized, the operative mortality is comparatively low.

Anatomically, intussusception consists of telescoping of an intestinal loop into another, distal, with ensuing damage to the blood vessels and

the wall of the telescoped loop. While in adults its occurrence can nearly always be traced to some anatomic abnormality, this is not the case in children, as in the latter nearly 95 per cent do not have any anatomic abnormalities.

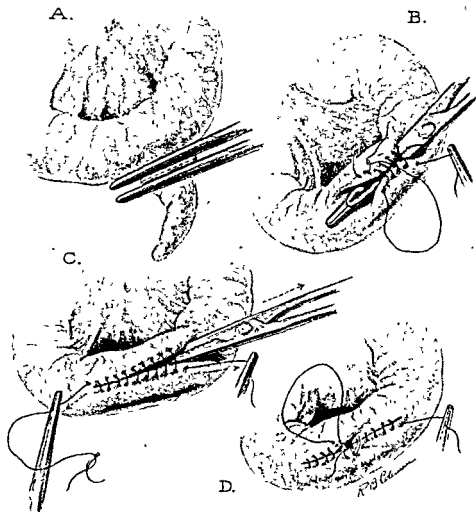


FIG. 222. Excision of Meckel's diverticulum. Management with aid of Kocher's clamps. A. Clamping of diverticulum with two clamps. Dotted line shows site of incision. B. Continuous over and over suture over clamp. C. Suture tightened gradually as clamp is removed. D. Second row of over and over sutures.

ETIOLOGY. This condition occurs more frequently in boys than in girls, the ratio being 3 to 2. More than fifty per cent of all cases of intussusception occur in infants between the 5th and 8th months. It usually

affects well nourished, healthy looking children, being seldom seen in thin, undernourished infants.

NOMENCLATURE. The condition of telescoping one loop into another is known as intussusception. The portion of bowel entering the other is

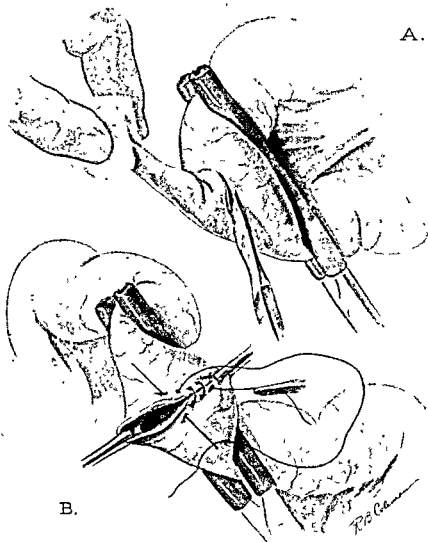


FIG. 223. Excision of Meckel's diverticulum by an open method. A. Excision of the diverticulum. B. Closure of the bowel in the direction perpendicular to the long axis of the bowel.

called intussusceptum, and the recipient loop is known as the intussusciens.

In seventy-five per cent of all cases, the terminal ileum telescopes into the cecum, forming the ileo-cecal variety. Other types, such as jejuno-jejunal, jejuno-ileal, ileo-ileal and colo-colic occur more rarely. Occa-

sionally, an intussusceptum with the intussusciens may telescope into a still more distal loop, thus forming more complicated types. This variety usually occurs in the terminal ileum and cecum, thus forming an ileo-ileo-cecal type. Extremely rarely the distal portion telescopes into the proximal thus forming a retrograde intussusception. Finally, one may occasionally find multiple intussusceptions.

PATHOLOGY. The entering bowel drags with it its mesentery which becomes stretched and compressed by the wall of the intussusciens. Secondary changes then occur in the wall of the bowel, to a greater degree of the intussusceptum and to a lesser of the intussusciens. These changes may be insignificant in early cases, but infarction and necrosis may occur in progressed cases.

SYMPTOMS. *Pain* is the first and outstanding symptom. It is colicky in nature, very severe, so that the little patient doubles up. It lasts ten or fifteen seconds. These attacks are separated by five to ten minute intervals, during which the child is seemingly well.

Vomitus is the next prominent symptom. It is severe and frequent. A few hours later pallor and dehydration become prominent; still later shock supervenes and the patient becomes moribund.

Melena is present in about eighty to eighty-five per cent either as dark brown mucus or as bright red blood which usually appears 12 to 24 hours after the onset. The amount of blood varies from a mere staining on the diaper to a copious flow.

Palpation of the abdomen reveals, in about eighty per cent, a sausage-like swelling which is soft and free from tenderness.

Rectal Examination reveals blood. Digital examination per rectum may palpate the approaching head of the intussusceptum in only about twenty-five per cent.

Bimanual Examination with a finger of one hand in the rectum and the other hand on the abdomen will detect the abdominal tumor, which by digital examination alone could escape detection.

Fever is absent at the onset; later, when dehydration takes place, there is a moderate elevation of temperature. With further progress of the condition, if infarcts or peritonitis occur, the temperature becomes high.

X-ray examination in nearly ninety-five per cent of the cases is unnecessary, as the clinical picture itself is sufficiently characteristic to enable one to make the diagnosis. As the greatest majority of cases involve some portion of the colon, roentgenologic study should be made by a barium enema, which gives quite a characteristic picture, such as a cupola shape of the barium where it meets the most advanced portion of the intussusceptum and a cylindrical shell of barium, surrounding the intussusception which remains after evacuation of the enema.

DIFFERENTIAL DIAGNOSIS. Intussusception should be differentiated from:

1. *Acute appendicitis*. This is very uncommon under the age of two. Fever and leukocytosis are present in appendicitis, but not in intussusception, appearing in the latter only when peritonitis is present. The onset in appendicitis is not as acute as in intussusception.

2. *Enterocolitis* does not have an acute onset. There are numerous watery stools with or without mucus or blood. Flatus is present; no masses are palpable.

3. *Prolapse of the rectum* does not cause abdominal pain, intestinal obstruction, palpable masses—symptoms present in intussusception. In case of prolapse, a finger cannot pass alongside the prolapsed bowel, while in intussusception the finger can be pushed into the rectum.

4. *Inflamed Meckel's diverticulum* resembles appendicitis. However, one should bear in mind that Meckel's diverticulum may occasionally be the starting point of intussusception.

5. *Henoch's purpura* may strikingly resemble intussusception. It may start with severe abdominal pain, the passage of blood through rectum, the presence of an abdominal mass due to subserous extravasation of blood along the bowel and often associated with shock. However, the purpura rash over the body, particularly on the extensor surfaces of the extremities, and a low platelet count differentiate the malady from intussusception.

TREATMENT. Two types of treatment are used to remedy this condition: I. Non-operative, and II. Operative.

I. *Non-operative* treatment was advocated by Hipsley. He reported several hundred cases of intussusception cured by enemas of saline solution given under pressure of 3 to 4 feet above the level of the patient's body in horizontal position. The technic is as follows: Under general anesthesia, a catheter No. 16, French scale, is placed into the rectum. One quart of saline solution held in a container 3 to 4 feet above the patient is allowed to run into the rectum. The buttocks are pressed to each other for three minutes so that no solution escapes outside. After this the solution is allowed to run outside and the same procedure is repeated several times.

As one may doubt whether the hydrostatic pressure relieved the obstruction, Hipsley gives the following signs of reduction:

1. Presence of abdominal distention after the enema was given is a sign that the water passed the point of intussusception with resulting reduction of the mass.
 2. Fecal material will flow with the returned saline solution.
 3. One teaspoonful of powdered charcoal mixed in half an ounce of water is administered by mouth. If the obstruction was reduced, the charcoal will pass through the rectum in five to seven hours.
- Other authors are using a barium enema under 2 to 3 feet pressure and observe the results by fluoroscopy. If the barium fills the entire length

of the large bowel and reaches the terminal ileum, the obstruction is reduced.

This simple treatment gives good results in cases of early intussusception. However, if more than 12 hours have elapsed since the onset the results are uncertain.

II. *Operative management.* This is resorted to by many surgeons from the very start and by others after medical treatment has failed. It consists of proper preoperative care, namely, of giving the infant glucose intravenously and by decompressing the stomach. The latter is a very important step, as it prevents aspiration of the gastric contents during anesthesia and facilitates manipulation in the abdomen after the peritoneal cavity is opened.

Anesthesia. Ether by open drop method is very efficacious. The abdomen is opened by a right paramedian incision four inches long, having the middle of the line at the level of the umbilicus. It is always preferable to have the incision on the right side even if the mass is palpable on the left side. The reason for it is that though the intussusceptum reached the transverse or even descending colon, in the greatest majority of cases it is of the ileo-colic variety. While the mass palpable on the left side is mobile and can be delivered outside the abdomen for reduction, it is not the case at the ileo-cecal junction; therefore, in this region we have to stay close to the cecum for which reason the incision should be on the right side. After the abdomen is opened, the next step, is the

Reduction of the Intussusception (Fig. 224). First we have to ascertain the most advancing point of the intussusceptum and then try from this point to milk out the intussusceptum in a proximal direction. It would be a grave error to start to look for the starting point and pull out the intussusceptum from intussusciens, as it is easy to tear both or the intussusceptum alone. In milking the advanced head, one has to be gentle. It requires patience until finally the telescoped loop is disengaged. Sometimes it may take a considerable time, from twenty to thirty minutes, but the effort is well worth trying, because the mortality is lessened if no resection is done. Under ordinary circumstances it takes about five minutes. Usually the reduction of the last few inches, close to the ileo-cecal junction is difficult.

Reduction is possible in ninety-five per cent of all cases. As soon as this is done, both loops, the intussusceptum and the intussusciens, should be carefully examined for change of color, gangrene of the wall or thrombosis of the mesenteric vessels. If the bowel wall is cyanotic but the pulsation of the mesenteric arteries is good, one should wrap the gut in question in a towel soaked in warm normal salt solution and leave it for five to ten minutes. If the color returns to normal, and if the bowel wall after being pinched by a tissue forceps shows peristaltic motion,



FIG. 224. Reduction of intussusception. 1. Abdominal incision. 2. Locating the most advanced point of the intussusceptum. 3. Milking out the intussusceptum in a proximal direction. 4. Reduction of the intussusceptum at the starting point.

the bowel wall is still good and it is safe to return it into the abdominal cavity.

If the serosa of the intussusciens shows tears, they may be closed by fine catgut sutures. If, however, one cannot reduce the intussusception or if after reduction one finds gangrenous bowel, no other course is left but to resect it. Several possibilities are open to the surgeon.

1. *Resection of the bowel with restitution of the continuity of the intestinal tract by end-to-end, or end-to-side, or side-to-side anastomosis, as a one stage operation.*
2. Lateral intestinal anastomosis between a loop proximal and another loop distal to the gangrenous bowel. The gangrenous bowel is exteriorized and removed a few days later and the two open ends are closed and placed into the peritoneal cavity.
3. Resection of the bowel by multiple stage operation of the Mikulicz type.
4. Ileostomy with exteriorization of the gangrenous bowel, the latter to be removed a few days later.

Some surgeons attempt to prevent a recurrence of intussusception by tacking down the terminal ileum to the posterior abdominal wall. This is a rather simple procedure and may be tried. However, experience shows that recurrence of intussusception even without this preventive measure is very rare; and those rare cases happened after such preventive surgery. For this reason we believe that this preventive measure has no particular value.

POSTOPERATIVE TREATMENT consists of parenteral administration of glucose or saline solution twice or three times daily, for two or three days following the operation. In the meantime the infants may be given only water by mouth on the first day and an equal amount of milk and whey on the second day. The normal amount of food is given on the fourth day.

MORTALITY. If the patient is left without treatment death is almost certain to ensue. Recovery by spontaneous reduction though possible is rare. This applies also to elimination of the gangrenous intussusceptum *per vias naturales*. Even after medical manipulations or surgical intervention the mortality is high. It depends on several factors of which the most important is the time elapsed between the onset of symptoms and surgical intervention. If surgery is resorted to within the first 48 hours, the prognosis is good. Ladd reported 110 cases operated within 48 hours without a single death, while in 92 cases operated between 48 and 96 hours, the mortality was thirty per cent.

Among the factors reducing mortality is proper preoperative and postoperative management.

Malformations of the Anus and Rectum (Figs. 225 and 226)

The statistics of these rare anomalies shows them to be present approximately once in every 750 new-borns. These malformations may be classed into three groups:

GROUP I. Stricture either of the anus or the lower rectum at some distance from the anus.

GROUP II. Absence of an anal opening. A membranous structure closes the lower end of the rectum. This group is encountered in nearly 70 per cent of all cases of malformation. In some, the lower rectum nearly reaches the external surface of the buttock, so that only a thin membranous layer is all that obstructs the outlet; in other cases the lower rectum may be at a considerable distance from the perineum, being as far as three or four centimeters from it.

GROUP III. The upper end of the anus and the lower portion of the rectum end blindly and are separated from each other at a variable distance.

In more than half of all these malformations there is a fistulous communication between the rectum and the bladder, urethra, perineum or vagina.

In male children the order of frequency of the fistulae are: rectoperineal (50%), rectovesical (33%) and rectourethral (17%).

In female children the order of frequency is: rectovaginal (85%), rectoperineal (14%) and rectovesical (1%).

The size of fistulae vary. In about forty per cent they are very small, in others they may be large enough to permit defecation through the bladder, urethra, perineum or vagina.

ASSOCIATED CONGENITAL ANOMALIES. In about 30 per cent of malformations of the rectum and anus one finds associated congenital malformations, such as heart anomalies, atresia of the esophagus, esophago-tracheal fistulae, atresia of the small or large intestine, Meckel's diverticulum, transposition of viscera, absence of a kidney, horseshoe kidney, exstrophy of the bladder, hypospadias, undescended testis, bicornuate uterus, atresia of the vagina, spina bifida, and the like.

THE SYMPTOMS vary depending on the type of the malformation, the presence or absence of fistulae, or their size.

In patients of group I, characterized by a varying degree of anal stricture, the infant has difficulty of defecation. In some cases this may force the patient's parents to call a physician's attention after a few days, while in other instances this may be unnoticed even for two or three years. The stool often has a "ribbon-like" appearance. Abdominal distention is usually present, but moderate. Vomitus is seldom present.

In cases of group II characterized by an imperforated anus, symptoms of intestinal obstruction appear within the first few days of life,

provided there are no associated fistulae of large size. Within 36 to 48 hours signs of intestinal obstruction, such as distention and vomitus,

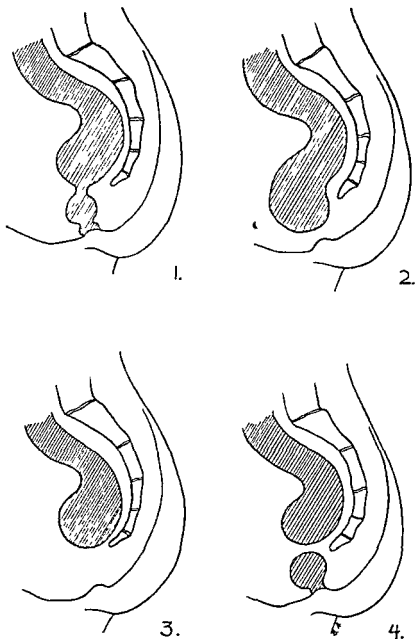


FIG. 225 Malformations of the anus and rectum. Type 1. Stricture of the anus. Type 2 Imperforate anus, close to the skin. Type 3. Imperforate anus, high in the pelvic floor. Type 4 Normal anus Rectal pouch ends blindly.

are already present. However, in about 60 per cent of this group there are associated fistulae of a large size permitting the feces to find an outlet. However, in this group the patient is brought to the physician the

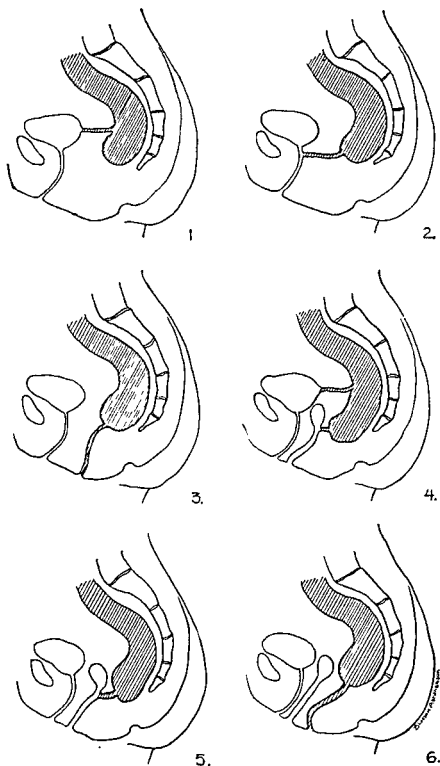


FIG. 226. Different types of fistulae between rectum, bladder, urethra and perineum. 1. Rectovesical fistula. 2. Rectourethral fistula. 3. Rectoperineal fistula. 4. Rectovesical and rectovaginal fistulae. 5. Rectovaginal fistula. 6. Rectoperineal fistula. — (After Ladd.)

second or third day of life, because there is no anal outlet.

Patients of group III show symptoms of intestinal obstruction 36 to 48 hours after birth, provided there are no associated fistulae of large size. The diagnosis in these cases is not easy, as the anus looks normal and one may think rather of atresia or of malrotation of the jejunum or ileum than of malformation of the rectum. However, if one thinks of a possibility of this variety, the diagnosis is not difficult as one will find that a small tube can enter through anus only for a very short distance, where it stops blindly and an x-ray examination made with the infant in the upside-down position (Wangensteen-Rice technic) shows gas bubbles in lowermost portion of the rectum.

THE TREATMENT varies according to the type of pathologic defect found and whether or not fistulae are present. In the type I cases uncomplicated by a fistula gradual dilatation with metallic or rubber dilators is all that is necessary. This dilatation is repeated daily for several weeks and later once or twice a week for several months. If a fistula is present the anus and rectum are freed from the surrounding structures, the fistulous tract is divided and the rectum is brought down so that the rectal opening of the fistula can be exteriorized.

Type II. In case of an imperforate anus in which only a thin membranous layer covers the lower rectum, a cruciate incision should be made through the imperforate anus, followed by daily dilatation with a metallic or rubber dilator. If, however, the rectal pouch does not lie immediately under the skin, the technic depends upon the distance of the pouch from the perineal surface. If the distance is not more than two centimeters, the operation may be done through the perineum. If, however, the distance is more than two centimeters, the operation should be carried out as a two-stage procedure. The first stage consists of a colostomy, followed in a few weeks or months by the second stage—the perineal approach to the pouch.

TECHNIC OF THE PERINEAL OPERATION. The patient is placed in the lithotomy position. A longitudinal incision is made along the perineal raphe, starting at the perineo-scrotal junction in males or from the vagino-perineal junction in females and reaching the tip of the coccyx. The incision divides the skin, superficial fascia and the external sphincter ani into two halves. The two lips are now reflected laterally exposing the levator ani muscles. The latter are separated from each other in the midline and the surgeon continues to dig deeper into the space normally occupied by the rectum until the blind pouch is reached. While working in this space to reach the blind pouch one should stay close to the hollow of the sacrum as otherwise one may inadvertently injure the urethra or the vagina. As a precaution one may insert a small catheter into the bladder or the vagina. When the rectal pouch is reached it is mobilized for some distance, so that it can be brought to the perineal surface by

moderate pull. Three or four traction sutures are placed through the end of the pouch penetrating only the seromuscular layer. These traction sutures are gently drawn until the distal end of the pouch appears outside the halves of the anal sphincter. The lips of the sphincter and of the skin are sutured to each other in front and behind the protruded pouch. The pouch is opened and its lips are now sutured to the skin. One should make sure that the rectal pouch is well beyond the anal opening so that when the rectum is sutured to the skin it will not retract by cutting through the sutures. If this will take place the space between the anus and retracted rectal pouch will constrict and there hardly will be a possibility of overcoming it by subsequent operations.

The operation is followed by daily dilatations for several weeks after which dilatation is done twice a week for several months.

If the malformation is of the type III, the surgical repair consists of a two-stage operation. The first is colostomy, and the second, which should be done weeks, months or even years later, consists of perineal exposure of the upper end of the lower pouch and the lower end of the upper pouch and uniting them by an end-to-end anastomosis.

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Chapter XV

Occlusion of the Mesenteric Vessels

By JULIUS L. SPIVACK

Occlusion of the mesenteric vessels is a subject treated meagerly in most textbooks of surgery. In many manuals it is omitted, while in others it is briefly discussed as one of the causative factors of intestinal obstruction. An explanation for this neglect is assumption that this condition is extremely rare, its diagnosis almost impossible and its treatment virtually hopeless.

However, in recent years considerable interest arose in this condition; numerous clinical and some experimental facts were presented; and although the prospects as to diagnosis and treatment are still not very favorable, they are far more hopeful than in the past.

HISTORICAL NOTE. Occlusion of the mesenteric vessels was reported by Hodgson, of Guy's Hospital in London, as early as in 1815. He described a case of compression of the celiac and superior mesenteric arteries by an aneurysm of the aorta. Fleishmann, in the same year, and Chaussier, in 1818, also described occlusion of the celiac and superior mesenteric arteries, and Meli, in 1821, reported occlusion of the superior mesenteric artery and vein. Tiedemann, in 1843, gave a clinical description of this disease, and Virchow, in 1847, recorded the morbid anatomy of mesenteric occlusion. Gerhardt, in 1863, published a series of ten cases collected from the literature and added one of his own. He gave the clinical description of this disease and formulated a group of postulates which should be demonstrated before diagnosing mesenteric occlusion. Litten, in 1875, presented an excellent paper on the subject. Since that time the subject remained dormant until Jackson, Porter and Quinby, in 1904, published a review of 214 collected cases. This report stimulated the interest of other investigators and many papers on the subject followed. Trotter, in 1913, published an excellent monograph in which he collected 359 cases which had been reported between 1847 and 1913, adding seven of his own. A. J. Cokkinis, in 1926, contributed a valuable monograph on the subject; in addition to many still unpublished clinical cases he described his original investigation on the distribution of the blood vessels in the bowel. Since that time many reports on this condition have appeared, some of single cases, others of group-series.

SURGICAL ANATOMY

Distribution of the Blood Vessels in the Bowel

The blood supply to the intestine, except the upper portion of the duodenum and the lowermost portion of the rectum, is derived from the superior and inferior mesenteric arteries.

The superior mesenteric artery and its branches supply the entire length of the small intestine with the exception of the proximal two-

thirds of the duodenum, as well as the appendix, cecum, colon ascendens, hepatic flexure, the right part of the transverse colon and, to some extent, the central third of the transverse colon. The inferior mesenteric artery supplies the rest of the large bowel with the exception of the lowermost portion of the rectum which is supplied by the inferior and middle hemorrhoidal arteries which originate from branches of the internal iliac artery.

The superior mesenteric artery has a constant point of origin and a fairly constant pattern of division of its branches and their inosculation, although the number of branches varies. This artery arises from the anterior wall of the abdominal aorta about one-half inch below the celiac axis. It enters the root of the mesentery and passes in front of the third portion of the duodenum; thus, the latter lies between the spinal column and the mesentery and, in some cases, its lumen may become obstructed. After entering the mesentery, the superior mesenteric artery runs obliquely downward and to the right forming a moderately curved line with its convexity toward the left; it is directed to the ileocecal angle but does not reach that far and ends approximately two inches from it by dividing into its terminal branches.

The branches of the superior mesenteric artery are: (1) **The inferior pancreatico-duodenal artery:** It supplies the distal portion of the duodenum and pancreas; it anastomoses with the superior pancreatico-duodenal artery and the first ramus intestini tenui. (2) **The middle colic artery:** It crosses the superior mesenteric vein and enters the transverse mesocolon. Here it divides into the right and left branches each of which unites with the ascending branch of the right and left colic arteries respectively, thus forming a single artery which runs parallel to the long axis of the transverse colon at a distance of one inch from its mesenteric border. From this artery short branches run perpendicularly to the wall of the bowel. (3) **The right colic artery:** This artery passes along the posterior abdominal wall to the right at the level of about the middle of the ascending colon, and at a distance of about one inch from the mesenteric border of this bowel divides into an ascending and descending branch which inosculate the former with the right branch of the middle colic and the latter with the colic branch of the ileocolic artery. (4) **The ileocolic artery** arises just below the middle of the root of the mesentery either by a common trunk with the right colic artery or just below it and runs to within one and a half inches of the ileocecal angle. It divides into ascending and descending branches; the former joins with the lower branch of the right colic and the latter with the colic, anterior ileocecal and posterior ileocecal branches of the terminal branches of the superior mesenteric artery. (5) **The terminal branches** of the superior mesenteric artery are ileal, appendicular, anterior ileocecal, posterior ileocecal and colic. (a) The ileal branch turns upward

and to the left and anastomoses with the lowermost *rami intestini tenui*. (b) The appendicular branch passes behind the terminal portion of the ileum, then enters the mesoappendix and reaches the appendix. (c) The anterior ileocecal artery crosses in front of the ileocecal junction and supplies blood to the anterior wall of the cecum. (d) The posterior ileocecal artery crosses behind the ileocecal junction and provides blood to the posterior wall of the cecum. (e) The colic artery runs upward along the ascending colon. (6) **Rami intestini tenui:** These arise from the left side of the superior mesenteric artery; they vary from ten to sixteen in number and furnish the blood supply to the entire length of the small bowel with the exception of the duodenum and the terminal few inches of the ileum. Their subdivisions and course have a definite pattern and are of great surgical significance.

Each artery divides into two branches which inosculate with the adjoining branches of the two neighboring *rami intestini tenui*, forming the first series of arcades. From these "primary" arches thin vessels pass to the wall of the jejunum—*vasa recti*. This arrangement is observed in the upper half of the jejunum. From this point distally to the beginning of the ileum another arrangement is observed, namely, the primary arches give off branches which divide and inosculate with the adjacent branches, thus forming "secondary arcades." In the upper half of the ileum, "primary," "secondary" and "tertiary" arcades can be seen and in the lower half of the ileum there is an irregular arrangement. *Vasa recti* pass from the last row of the "arches" to the wall of the bowel on which they ramify. These *vasa recti* are longest in the upper part of the jejunum, reaching one and one-half inches in length; and they are *shortest in the terminal ileum where their length is only one-half inch*. The *vasa recti* run alternately on the anterior and posterior wall of the bowel and then ramify. Occasionally, the branches of one *vasum* anastomoses with the terminal branches of the adjoining *vasa*. However, as a rule the *vasa recti* do not anastomose, which is of great surgical significance, owing to the fact that should the terminal arch become occluded by an embolus or thrombus on both sides of the arch, the wall of the gut along this arch would become gangrenous.

The inferior mesenteric artery arises from the front and left side of the abdominal aorta at a distance of one-half to two inches above its bifurcation. It gives off the following branches: (1) The left colic artery which runs laterally to within one to two inches of the middle of the descending colon where it divides into an ascending and descending branch. These branches inosculate with the left branch of the middle colic artery and the ascending branch of the first sigmoid artery, respectively. (2) The sigmoid arteries, two to four in number, pass into the pelvic mesocolon and each is divided into an ascending and descending branch which inosculate each ascending branch of the lower sigmoid

with the descending branch of the higher sigmoid, thus forming an artery which runs parallel to the descending and sigmoid colon and which is known as the marginal artery. (3) The superior hemorrhoidal artery, which is the terminal branch of inferior mesenteric artery.

ETIOLOGY. INCIDENCE. Occlusion of mesenteric vessels is rare as is shown by the fact that in the Charity Hospital of New Orleans it was encountered only thirteen times out of 300,000 admissions; in the Emergency Hospital, Washington, D. C., in 30,000 admissions only seven times; in the Boston City Hospital in thirty-three years only fifty-one cases were seen; in the Massachusetts General Hospital in 50,000 surgical admissions only thirteen times. Reports from other hospitals show about the same ratio.

AGE. It is a disease of middle age or of elderly individuals, although cases have been reported in children. The youngest case on record is that of an infant four months old (in J. L. Meyer's series) and the oldest ninety years old (in the Jackson, Porter and Quinby series). In Trotter's series 63 per cent of all patients were under the age of fifty.

SEX. There is a preponderance of the male over the female sex at a ratio of 2 to 1.

RACE. There is a decided preponderance in whites over negroes, 3 to 1.

PATHOLOGY. The mesenteric vessels may be occluded in several ways: (1) By embolism and thrombosis; (2) by inflammatory or degenerative changes in the wall of the vessels which occlude the lumen; (3) by external pressure and (4) by trauma.

Occlusion by Embolism and Thrombosis. An occlusion may be arterial, venous or combined, the latter variety affecting both the artery and vein. According to early investigators, arterial occlusion occurs more frequently than the venous type. In Trotter's series of 366 cases arterial occlusion occurred in 53 per cent; venous in 41, and combined in 6 per cent. However, more recent reports claim that venous occlusion is more frequent. Thus, in Cokkinis' series of seventy-six cases, 75 per cent were venous and 25 per cent arterial; in Brady's report from Johns Hopkins Hospital eight out of fourteen cases were venous, four arterial, and in two cases Brady could not determine the exact nature. L. D. Whittaker and John de J. Pemberton reported sixty cases from the Mayo Clinic in which arterial occlusion occurred in 31.7 per cent, venous in 45, and the combined form in 23.3 per cent.

Arterial occlusion may be due to embolism or thrombosis. In most cases (almost 90 per cent) it is caused by embolism and only in 10 per cent by thrombosis. The emboli set out from the left half of the heart, the aorta, or the lungs. When they come from the heart they may be attributed to some pathologic changes in it, such as acute or chronic endocarditis. When they arise from the aorta, the emboli usually originate in the aortic valves and, in a few instances, in the atheromatously

changed wall of the aorta. Venous occlusion is usually due to thrombosis and very seldom to embolism.

Primary and Secondary Thrombosis. Thrombosis may be primary or secondary. Primary venous thrombosis is much rarer than secondary. In Cokkinis' series of fifty-six cases of venous occlusion, the primary type occurred in only four cases. Secondary thrombosis accounts for the greatest majority of cases of venous occlusion. It is secondary to pathologic changes in the portal vein, or the intestinal wall, which is frequently a lesion of the appendix. Other causes of thrombosis of the mesenteric vein are diverticulum of the sigmoid, excision of the rectum, ulcerative colitis, purulent endometritis and salpingitis. It also occurs after hemorrhoidectomy, perineorrhaphy, Kraske operation, strangulated hernia, hepatic abscess and volvulus.

Inflammatory Changes. Inflammatory or degenerative changes in the wall of the vessels are a very rare cause of occlusion and only a few cases are recorded. The actual condition is *endarteritis obliterans*, and *endo-phlebitis obliterans*.

External Pressure. Occlusion of the superior or inferior mesenteric arteries by outside pressure has been reported by several authors. In most cases pressure is exerted by an aneurysm of the abdominal aorta upon the mesenteric vessels in the part which passes along the wall of the abdominal aorta. The occlusion is in some cases partial, in others complete. It is significant that in some cases of complete obliteration of the lumen of the artery, the gut is not gangrenous. This can be explained by the fact that the obliteration was gradual and collateral circulation developed to a sufficient degree. A strangulated hernia may constrict the mesenteric vessels or its branches. A case was recently seen by the author when he was called in consultation to see a middle-aged woman who complained chiefly of pain in the chest and of some abdominal distress of three days' duration. Physical examination at that time revealed a strangulated femoral hernia. Under spinal analgesia the sac was opened and the exposed loop of the bowel was cyanotic. After incising the ring to attain release of the bowel, it was found that the mesentery of the loop was entirely thrombosed to its roots.

Trauma. This is a rare occurrence in this altogether rare entity. In such cases the abdomen is injured by an external force such as being run over by a vehicle or similar accident. The mesenteric blood vessels are torn and blood, filling the space between the leaflets of the mesentery, compresses the blood vessels leading to the bowel with resultant gangrene. In this respect trauma actually produces a condition discussed in the preceding paragraph on External Pressure.

The superior mesenteric artery is affected far more frequently than the inferior. In rare cases both the superior and inferior mesenteric arteries are involved. The superior mesenteric artery alone is affected almost

twenty-five times as often as the superior and inferior combined, and fifty times as frequently as the inferior mesenteric artery alone. There are several reasons why the superior mesenteric artery is occluded by emboli more frequently than the inferior. The main reason for it is that it lies considerably higher than the inferior mesenteric artery and is, therefore, the first where an embolus can become lodged.

The pathologic picture varies according to the occlusion being arterial, venous or combined, gradual or sudden, and also depending on the site of occlusion—at the beginning of the artery or in some of its branches. Arterial occlusion usually is due to embolism and has a sudden onset. In high embolism, when the embolus lodges high in the superior mesenteric artery above the origin of its branches, there is a sudden cessation of the circulation of blood in the entire mesentery. The blood below the embolus empties into the arterioles and capillaries; necropsies have shown the artery below the embolus and its branches to be nearly empty, whereas the arterioles and the capillaries are filled with blood. If, however, embolic occlusion occurs distally to any of the large branches, be it only one and one-half inches below the origin of the superior mesenteric artery, the pathologic picture is different; the blood flows through the branches originating proximally to the point of occlusion and, through the collateral circulation, reaches the portion of the gut supplied by the occluded arteries and, thereby, does not damage the wall of the intestine. In some cases secondary thrombosis develops at the points of anastomosis of the arterioles of occluded and non-occluded arteries and for this reason infarction of the gut still will take place. If, however, no secondary thrombosis develops, the gut in this low type of embolus may remain undamaged. The damage to the wall of the intestine further depends upon the embolus occluding an arterial branch close to or far from the intestinal wall. If the embolus lodges in the vasa recti, there will be damage to the wall of the gut supplied by the occluded vasa recti because there is usually no anastomosis between them. If, however, the embolus blocks the main branch of the artery or the proximal arcade, the presence or absence of thrombosis decides whether or not there will be damage to the intestinal wall.

In venous occlusion the picture is different. As it is usually due to thrombosis, the occlusion proceeds gradually and, therefore, also the development of a collateral circulation. Owing to the thinness of the walls of the veins small venous collateral branches become distended and may form large trunks. Cases have been recorded in which the vena porta, the superior or inferior mesenteric veins were completely occluded for several years without any clinical signs because of the development of a compensatory collateral circulation. However, if the thrombosis spreads to the last venous arcades or the vasa recti, the collateral drainage is blocked, the vein can not empty, the arteries are still carrying

blood, the venous capillaries become engorged and, finally, torn, producing hemorrhagic infarction. The appearance of the abdominal cavity in mesenteric thrombosis is characteristic, and an excellent description of it was given by R. G. Loop: "Transparent, sticky peritoneal fluid amber or blood-tinged, and without coagulated lymph. Cyanosed, plum colored soggy edematous intestine with glistening peritoneum, free from adhesions, its lumen relaxed to a large caliber, lying inert in the abdominal cavity with no tendency to crowd out of the incision, held down by the weight of the fluid within its lumen, containing very little gas."

SYMPTOMS. The symptoms depend upon many factors, such as arterial or venous occlusion, produced by an embolus or a thrombus, whether the superior or inferior mesenteric artery is affected; whether the point of occlusion is high, i.e., at a point of the main stem lying higher than its branches, or low, i.e., below the point of origin of at least one branch, whether one or more arcades and any vasa recti are occluded, whether occlusion of the vessels is the only pathologic process or whether it is accompanied by endocarditis, pylephlebitis or appendicitis. According to the cause and character of constriction, the symptoms may appear suddenly or insiduously; and as an acute appearance is more readily observed, this variety was first described, usually associated with mesenteric occlusion.

From the standpoint of clinical manifestations mesenteric occlusion may be classed into three groups: (1) Acute, (2) chronic and (3) latent.

Acute Group. In a typical case the first symptoms are: (1) **Pain:** It appears suddenly, is colicky and very severe; later it becomes continuous and it is, usually, over the entire abdomen. Sometimes it is referred to the epigastric or right lower quadrant; occasionally, it is felt in one or the other loin. However, even if it is diffuse, it is still most marked at the seat of the pathologic process. It is the most persistent symptom and is present in more than 90 per cent of all cases. (2) **Vomiting:** This is the next symptom in the order of frequency and appearance. It is present in about 75 per cent of all cases; the patient vomits once or twice and then the vomiting ceases, but recurs later with other symptoms of peritonitis. (3) **Constipation, obstipation or diarrhea** is present in about 55 per cent and constipation and obstipation are more frequent than diarrhea. In some cases the stool is regular and in others constipation or obstipation follows diarrhea and still in others diarrhea follows constipation. In Trotter's series of 366 cases irregular function of the bowels was recorded in 209 cases; constipation or obstipation in 103 cases, diarrhea in eighty-seven; constipation or obstipation followed diarrhea in fifteen and diarrhea followed constipation in four cases. (4) **Melena:** The frequency of this symptom varies considerably. In Cokinis' series melena was reported in 18 per cent; in the statistical review of Jackson, Porter and Quinby of 214 cases, melena was present in 31 per

cent. (5) Hematemesis occurred in about 28 per cent of all cases of Trotter's series and in about 25 per cent of Cokkinis' series. The blood is dark brown or black, in some cases bright red. It is clinically significant because, like melena, it is a valuable guide in differentiating between a simple intestinal obstruction and that due to mesenteric thrombosis. (6) Shock is present in nearly all cases with an acute onset. (7) Symptoms of internal hemorrhage, such as restlessness, intense thirst, faintness and air-hunger are frequently present. (8) Collapse often takes place. It is due to exhaustion from continuous severe pain, from loss of blood by melena, hematemesis and extravasation into the peritoneal cavity, to dehydration by vomiting and diarrhea and to toxic absorption from the gangrenous bowel. The patient is pale and looks extremely ill; the expression of his face is anxious (Hippocratic.) (9) Distention of the abdomen occurs early and is general; occasionally, it is limited to the epigastrium or hypogastrium. It occurred in 65 per cent of all cases of Cokkinis' series. Although it is present in arterial and venous occlusion, it is more pronounced in the latter. (10) The temperature is subnormal. Occasionally, it rises slightly above normal, but then drops to subnormal. This marked fall, after a slight rise, is a characteristic feature. If the temperature remains elevated, it is usually due to the etiologic factor, or to an associated condition such as endocarditis, pyemia, appendicitis, and the like. (11) The pulse is rapid from the start and becomes increasingly faster. It may reach 160 and even become uncountable. It is usually small, irregular and thready. (12) Free fluid in the abdomen is a very valuable sign. This is one of the most frequent symptoms of mesenteric thrombosis and occurs in about 85 per cent of all cases. It is manifested by shifting dullness in the flanks. (13) Rigidity is present in about 45 and tenderness in about 65 per cent. These symptoms may be generalized or confined to one or another region, such as the epigastrium, hypogastrium, right iliac or umbilical region. Rigidity is present in cases in which peritonitis already has set in. If it is absent, it is in striking contrast to the subjective sign of pain, which is extreme in its intensity. (14) Leukocytosis reaches a high level, up to 30,000 and more. It is higher than in any other acute abdominal condition. The increase is rapid and occurs soon after the onset of pain.

Characteristic for this disease are the severe pain, high leukocyte count and subnormal, normal or only slightly elevated temperature ranging between 96° and 101° F. These symptoms are present only in a typical case of acute mesenteric arterial occlusion. However, in some cases the symptoms are different.

Chronic Group. This group is characterized by a chronic course of the disease which ultimately becomes acute. The patient complains of symptoms which extend over weeks or even months, such as colicky abdominal pain, vomiting, diarrhea followed by constipation or vice versa, in-

creased abdominal distention and flatulence; later, attacks of hematemesis and melena. These symptoms disappear for several weeks or months and the patient seems to be in comparatively good health until there is a sudden attack of the acute obstructive or hemorrhagic type of occlusion and the patient is extremely ill. This chronic variety of mesenteric occlusion occurred in 21 per cent of Cokkinis' series. In its chronic phase it is readily mistaken for duodenal ulcer, appendicitis and carcinoma of the colon; when the acute symptoms appear they may be mistaken for a perforated viscus or peritonitis.

Latent Group. In this group the mesenteric occlusion is not a prominent feature and may even be overlooked. If, for instance, the patient suffers from pyemia with emboli disseminating in different arteries, some of them may lodge in the rami intestini tenui without producing gangrene or intestinal obstruction. The patient succumbs to pyemia and it is only at autopsy that emboli are found in the mesenteric arteries.

DIAGNOSIS. The diagnosis is difficult, particularly in the chronic type or when mesenteric occlusion is a sequel to some other condition, the symptoms of which are more pronounced. That is why in most of the cases the diagnosis is made only after the abdomen is opened. A correct diagnosis is made in only about 15 per cent.

In order to diagnose the acute type, the surgeon should bear in mind the following: (1) A history of a causative lesion, such as acute endocarditis, pyemia, disease of the valves of the heart, portal obstruction or vascular occlusion in other regions. (2) A sudden onset of severe colicky pain, early vomiting, diarrhea or constipation, subnormal temperature, rapid, thready pulse which increases hourly in frequency, high leukocytosis, signs of external or internal intestinal hemorrhage and frequently intestinal obstruction. (3) An enema which reveals blood is the most valuable single symptom of mesenteric occlusion. (4) Constant drop in the blood pressure taken at short intervals.

DIFFERENTIAL DIAGNOSIS. Many abdominal conditions may be confused with acute mesenteric occlusion: (1) Certain types of acute intestinal obstruction; (2) perforation of hollow abdominal viscera, such as the stomach, duodenum or appendix; (3) hemorrhage from a gastric or duodenal ulcer and (4) acute pancreatitis.

For differentiation from acute intestinal obstruction it should be remembered that (1) acute intussusception affects infants and small children; (2) chronic intussusception usually affects adults and does not present the acute symptoms of mesenteric occlusion; (3) strangulation by a band presents strikingly similar symptoms; however, the occurrence of diarrhea, melena, or hematemesis, sanguineous results from an enema and a history or signs of a causative lesion or of vascular occlusion elsewhere speak for mesenteric occlusion. (4) Volvulus manifests vomiting as a late symptom, whereas in mesenteric occlusion it is an early

symptom; tenesmus is a frequent symptom of volvulus and rarely is observed in mesenteric occlusion.

In perforation of a hollow viscus with ensuing peritonitis the symptoms may closely resemble those of mesenteric occlusion; however, the differentiation is not difficult because in mesenteric occlusion there is colicky pain, early distention of the abdomen, signs of hemorrhage, slight rigidity, while in peritonitis due to rupture, there is extreme rigidity, immobility, tenderness, scaphoid retraction of the abdomen and a rise in the temperature.

Hemorrhage from a gastric or duodenal ulcer is usually not associated with severe abdominal pain or symptoms of intestinal obstruction.

Acute pancreatitis strikingly resembles mesenteric occlusion. A diagnostic enema shows blood in mesenteric occlusion, which renders possible the differentiation from acute pancreatitis.

TREATMENT. The treatment of mesenteric occlusion is surgical. The shorter the time between the onset of the disease and operation and the smaller area of gut involved, the greater the chances for recovery. Despite early and skillful operation, the mortality rate is appalling, being as high as 80 to 85 per cent; under medical treatment the mortality reaches nearly 100 per cent. Klein, in 1921, found in the entire literature only eight cases of spontaneous recovery. Resection of the bowel is the most logical operative procedure. The first successful enterectomy for this condition was performed by J. W. Elliot in 1894. The size of the removed bowel varied from a few inches to fifteen feet or almost two-thirds of the length of the small bowel. In the course of time better results have been obtained; Brady, in 1923, had three recoveries in eight operated patients, which is one of the best reports on record.

The next logical procedure is exteriorization of the affected bowel with a portion of healthy bowel lying proximally and distally. In the normal proximal portion of the bowel a tube is inserted for drainage. It is always advisable to exteriorize at least one foot of normal bowel on each side, provided its combined length with the gangrenous bowel does not exceed one-half of the length of the small bowel. The reason for this is that in many cases the line of demarcation between the healthy and diseased portion is not sharp and a seemingly healthy part of the bowel may become involved within a few hours.

Enterostomy proximal to the gangrenous loop of the bowel, leaving the impaired bowel in the abdomen, was carried out by several surgeons. This operation does not accomplish much; although it drains the intestinal contents outside, it leaves behind the gangrenous part which serves as a source of infection, intoxication and peritonitis. We consider it a futile procedure. The technic of enterectomy, exteriorization and enterostomy is described elsewhere.

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Chapter XVI

Urgent Surgery of the Colon

By JULIUS L. SPIVACK

Carcinoma

Carcinoma of the intestinal tract in most cases does not belong to the domain of urgent surgery. However, its complications, such as acute intestinal obstruction and perforation, certainly present urgent conditions and for this reason carcinoma of the bowel becomes a subject of urgent surgery.

Carcinoma of the intestinal tract is most often confined to the colon, its occurrence in the small bowel being in only about one per cent of the cases. In the large bowel not all parts are affected with equal frequency: the rectosigmoid and rectum are affected in about 64 per cent and the rest in 36 per cent. Eighty per cent of carcinomas of this region can be palpated by a finger introduced into rectum.

ETIOLOGY. Carcinoma of the large intestine represents about 15 per cent, while cancer of the entire gastro-intestinal tract represents about 48 per cent of all cancers of the body. In different parts of the large bowel the distribution of cancer is as follows: cecum 15 per cent, transverse colon 10 per cent, splenic flexure 6 per cent, descending colon 5 per cent, rectosigmoid and rectum 64 per cent.

AGE. Most frequently it affects individuals in the 5th and 6th decades; however, cases have been recorded even in children of the age of five and seven. Pennington in a series of 7,313 cases of carcinoma of the colon reported 40 cases under the age of 20; 235 from twenty to thirty years; 690 from thirty to forty; 1462 from forty-one to fifty; 2120 from fifty-one to sixty; 1836 from sixty to seventy; and 930 cases above the age of seventy.

RACE. Whites are considerably more often affected than negroes.

Males are more frequently affected than females, the ratio being 2 to 1.

PATHOLOGY. Carcinoma of the colon is usually primary and presents a single focus; however, multiple foci are encountered. Histologically, it is divided into the following groups:

- a. Medullary adenocarcinoma;
- b. Scirrhous carcinoma, or fibrocarcinoma;
- c. Colloid (mucoid) adenocarcinoma;
- d. Papillomatous carcinoma;
- e. Squamous carcinoma; and
- f. Melanoma,

Different types of carcinoma are responsible for marked variations in the symptoms as well as the roentgenologic picture of the affected bowel. However, regardless of the pathologic type, they all may ulcerate with ensuing secondary infection. This infection is first localized in the ulcerated carcinomatous mass, and later may spread in continuity, suppurate and break outside, producing fecal fistulae, or into the free peritoneal cavity causing fatal peritonitis, or becoming walled off by adhesions and then forming intraperitoneal abscesses.

Carcinoma is usually single; multiple primary carcinoma of the colon is found only in about 3 per cent of all cases.

Metastasis in carcinoma of the colon occurs comparatively late, later in fact than in any other portion of the gastrointestinal tract. For this reason radical surgery in early cases of carcinoma of the large bowel offers a greater hope for radical cure than in any other organs affected by cancer. And of all parts of the large bowel metastatic carcinoma from the cecum occurs the latest.

The grade of malignancy has a direct bearing on the percentage of metastasis, since it increases in proportion to the grade.

In the order of decreased frequency metastasis occurs in the rectum, cecum, sigmoid, descending colon, transverse colon, splenic flexure and ascending colon. Metastases are usually located in the neighboring lymphatic glands.

As regards distant metastases the liver is the organ that is first affected.

SYMPTOMATOLOGY. The symptoms vary, depending on the site of the lesion (right or left colon), pathologic type, presence of complications, such as metastasis, ulceration and perforation.

Change of intestinal habit takes place in about 80 per cent of the cases of carcinoma of the bowel. Irrespective of its site, patients have diarrhea which is followed by constipation or vice versa. Therefore, in all cases of a sudden change of intestinal habit in people past middle age, the physician should not overlook the possibility of a malignancy.

Blood in the stool may be fresh or occult. In the latter form it can be found in the greatest majority of cases irrespective of the site of the lesion. However, fresh blood occurs with more frequency the closer the lesion is to the anus. Thus, in carcinoma of the cecum, it occurs in about 9 per cent, in carcinoma of the left colon in 46 per cent, and in carcinoma of the rectum in about 85 per cent.

Abdominal cramps occur in about 80 per cent in cases of carcinoma of the ascending or descending colon and only in about 10 per cent in cases of carcinoma of the rectum. If the lesion is in the right colon, it is often associated with tenderness in the same region, which accounts why from time to time a diagnosis of chronic appendicitis or cholecystitis is erroneously made.

Secondary anemia may be present in carcinoma of any portion of the large bowel. However, it is an outstanding feature in carcinoma of the cecum or of the ascending colon. The patient's face acquires a characteristic lemon-yellow tint. In no other place does carcinoma give such a characteristic appearance of the face as in that of the right colon. Whereas anemia of this type in carcinoma of the left colon appears only in far advanced, virtually inoperable cases, in case of carcinoma of the cecum it may appear in a comparatively early stage of the disease. Accordingly, anemia associated with carcinoma of the left colon is actually a contraindication to radical surgery, whereas with carcinoma of the right colon it does not constitute a contraindication.

Loss of weight is more pronounced in malignancy of the cecum and ascending colon than in the left colon, the reason for it being that the right colon has a great deal to do with absorption of liquid intestinal contents, while the left colon is chiefly a place of storage of feces. Another reason is that the intestinal lumen is widest in the cecum and the contents are liquid, for which reason the carcinomatous process may last for a considerable time, the patient may lose much weight and become dehydrated and still not have pain or become obstructed, the two symptoms that usually bring the patient to the physician.

Obstructive symptoms appear in about 25 per cent of all cases of carcinoma of the left colon and considerably less frequently in carcinoma of the right colon or cecum. In the latter case they appear in the late stage of the disease, but may manifest themselves in early stage when the tumor affects the cecal wall close to the ileocecal valve.

Obstruction may be *acute, subacute or chronic*. Acute obstruction is due to occlusion of an already narrowed lumen by fecal masses, volvulus or intussusception.

In *chronic obstruction* the patient becomes progressively constipated with signs of visible peristalsis, borborygmi and increased flatulence.

Palpation through the abdomen of a lesion of the cecum or ascending colon is not easy, particularly in the early stage of the disease or in a stout person; palpation of the hepatic flexure is still more difficult, due to its deeper position. However, palpation of the sigmoid is not difficult and particularly digital examination per rectum will reveal the presence of a tumor in these parts in about 80 per cent.

X-ray findings in the cecum and ascending colon are well pronounced in the later stages of the disease but may be overlooked in the early stage. Therefore, if x-ray findings do not corroborate the clinical findings it is advisable to take another x-ray 4 or 6 weeks later. Barium enemas should be given preference to oral administration, as the barium administered orally may plug the narrow lumen of the bowel and produce acute intestinal obstruction. Proctoscopic and sigmoidoscopic visualization is very important for the diagnosis. This may reveal not only the

characteristics of the growth, such as ulcerations or a papillary adenomatous lesion, but also will show the size, mobility and degree of obstruction of the bowel.

DIAGNOSIS. Loss of weight in persons past middle age, loss of strength, change of bowel habit, secondary anemia, occult or bright blood in the feces, a palpable mass felt through the abdomen or by finger introduced through the anus and a filling defect demonstrated in a roentgenogram and proctoscopic examination establish the diagnosis.

However, in many cases some of these symptoms are missing and even x-ray examination, particularly of the descending colon, may prove negative, especially so in the early stage of carcinoma. On the other hand, secondary anemia without any visible causes, particularly in the presence of a lemon-color tinge of the face in the middle aged, should always cause us to think of the possibility of carcinoma of the cecum or ascending colon.

DIFFERENTIAL DIAGNOSIS. This should be made from diverticulosis, polyposis, regional ileitis, chronic, non-specific granuloma, ulcerative colitis, actinomycosis, retrocecal appendicitis, bleeding hemorrhoids, benign strictures and intussusception.

Diverticulosis is readily diagnosed roentgenologically. The diverticula have an appearance of rounded, knob-like projections along the lumen of the bowel and are multiple. The filling defect observed in diverticulitis is of spastic origin and antispasmodic drugs, administered until the physiologic effect is obtained, will change the appearance of the bowel, while in carcinoma no change in the picture is observed after the administration of antispasmodics.

Polyposis. This occurs usually in younger persons under the age of 30; is present in many members of the same family; and x-rays by the "combined double contrast" method described by Fisher give a quite characteristic picture of polyposis.

Hyperplastic tuberculosis is usually localized at the ileocecal junction. The differential diagnosis from carcinoma is made chiefly roentgenologically since in tuberculosis the x-ray shows involvement of the terminal ileum and irritability around the affected segment.

Nonspecific granuloma may very closely resemble carcinoma both in its clinical manifestations and in the gross appearance of the lesion. It occurs mostly in the ascending colon. It presents great differential diagnostic difficulties, as is attested by the fact that in 90 per cent of the cases it was diagnosed preoperatively as "tumor." The fact that these patients are not anemic and have a general feeling of well-being shows that the patient has no carcinoma.

Chronic ulcerative colitis is differentiated by the onset, clinical course and particularly proctoscopic examination, which gives quite a characteristic picture both in the acute and the chronic stage of the disease.

In the acute stage hyperemia, edema, miliary abscesses and miliary ulcers are observed. In the chronic stage one sees contracted scars and a pitted mucosa. Granular ulcerations, punched scars and contraction of the lumen of the bowel are pathognomonic proctoscopic signs.

Actinomyces usually (in 80 per cent) affects the ileocecal region. It starts under the guise of acute or subacute appendicitis and later abscesses, sinuses and leathery infiltration of the surrounding tissues take place. The presence of sulphur granules after the formation of sinuses or microscopic examination of the affected tissues establish the diagnosis.

TREATMENT. There are few malignant growths which offer such a good prospect for radical cure as carcinoma of the large bowel, provided operation is done in the early stage of the disease before metastasis and general debility take place. Depending on the extent of the involvement surgery may be either radical or palliative. In the former the affected bowel is resected with all involved lymph glands. In palliative surgery only some type of side tracking operation or a colostomy is carried out.

Whether radical or palliative surgery is done depends on many factors: mechanical resectability—that is whether the affected bowel is freely movable or penetrates the surrounding tissues; extent of lymphatic involvement, presence of distant metastases, involvement of vital neighboring organs in continuity and the general condition of the patient, i.e. age, weight, cardio-renal factors and degree of anemia. Anemia is not a contraindication to radical operation, but necessitates an operation in several stages, which otherwise would be performed in one stage.

Distant metastasis contraindicates radical surgery. If the carcinoma of the colon has invaded the anterior abdominal wall, removal of the bowel with resection of a portion of the abdominal wall (laparectomy) is advisable.

Obese patients are poor risks owing to easier development of wound infection; age is an important factor because the older the patient, the more dangerous are the immediate results of operation; while the younger the patient, the more unfavorable is the prognosis with regard to recurrence. Anemia accompanying carcinoma of the left colon is not as dangerous from the operative point of view as when associated with carcinoma of the right colon because anemia in the former may be present in the early stage of the disease while in the latter it appears in the terminal stage.

Preoperative management consists of combating dehydration by sufficient amounts of glucose given subcutaneously (5 per cent) or intravenously (10 per cent) and blood transfusions to raise the hemoglobin level to 70 per cent. Decompression of the bowel is of greatest importance and, last but not the least, administration of sulfasuxidine (succinylsulfathiazole) diminishes the *B. coli* flora and reduces the occurrence of postoperative peritonitis. The amount of sulfasuxidine to be given is

0.25 gm. per kilo of the body weight daily. It can be given in 3 equal doses. It should be started five days before the operation, so that by the time of operation, the coli bacilli are greatly reduced in number.

CHOICE OF OPERATION. The operative procedures differ depending on whether the right or the left colon is involved. In cancer of the left colon the technic differs depending on whether we wish to preserve the sphincter ani or not.

The extent of operative removal for lesions at different sites of the colon is shown in accompanying figure (Fig. 227).

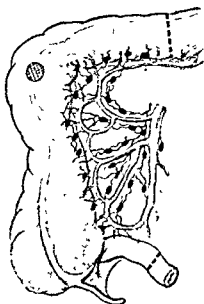
A. SURGERY OF THE RIGHT COLON (Figs. 228, 229, 230, 231). In malignancy of any part of the right colon (appendix, cecum, ascending colon, hepatic flexure, right third of the transverse colon), the entire right colon should be removed, together with 8 inches of the terminal ileum. This can be done as a one or a two stage operation.

In the one stage operation, the terminal ileum is divided at a distance of 8 to 10 inches from the ileocecal junction and the terminal portion of ileum, cecum, ascending colon, hepatic flexure and right third of the transverse colon are removed. Continuity of the intestinal tract is reestablished by suturing the terminal ileum to the remaining portion of the transverse colon. This can be carried out as end-to-end, end-to-side or side-to-side anastomosis. Of course in side-to-side anastomosis the open ends of the ileum and of the transverse colon are closed and in end-to-side anastomosis the open end of the transverse colon is closed. The actual anastomosis can be done by the open or closed method.

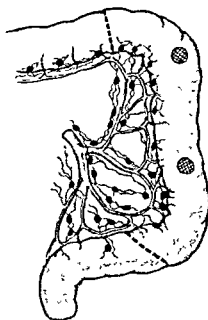
The technic of the closed clamp one-stage method is as follows: The abdomen is opened by a right pararectus incision starting 2 inches below the costal arch and extending downward 5 to 6 inches. The terminal portion of the ileum is divided between two crushing clamps, placed in a slightly oblique position so as to increase the lumen of the bowel and to secure a better blood supply to its cut edge. A fold on the anterior wall of the transverse colon is grasped by another clamp and the fold is shaved off flush to the blade. The length of the removed fold should be equal to the size of the lumen of the proximal cut end of the ileum. The clamp holding the proximal end of the divided ileum is placed side by side with the clamp holding the transverse colon so that the two openings lie symmetrically side by side. Sutures are placed uniting the posterior and the anterior lips of the two openings, thus uniting ileum with the colon. The clamps are now opened and gradually withdrawn by the surgeon while the assistant draws in opposite direction the ends of the anterior and posterior sutures, thus inverting the lips of the two segments of bowel. The corresponding ends of each thread are tied to each other, care being taken not to occlude the newly established anastomosis by a purse-string action. A second layer of seromuscular-seromuscular suture reinforces the first layer. The anastomotic opening is



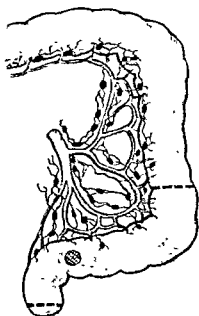
A



B



C



D

FIG. 227. Extent of colon resection depending on the site of pathology. A. Carcinoma of the cecum. B. Carcinoma of the hepatic flexure. C. Carcinoma of the descending colon. D. Carcinoma of the sigmoid.

now made permeable between the index finger and the thumb. Next, the parietal peritoneum, lateral to the cecum and ascending colon is cut and the inner peritoneal lip is grasped. A finger is insinuated behind the

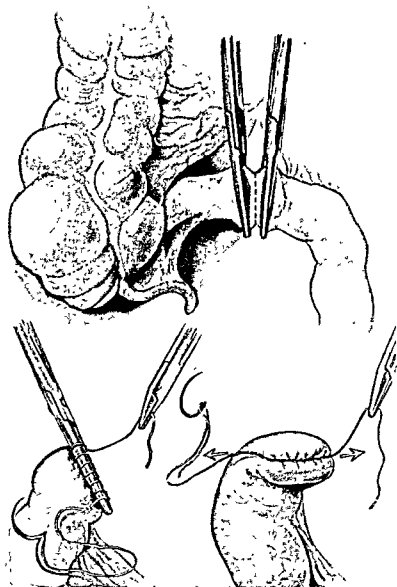


FIG. 228. Right hemicolectomy. Upper half. Cutting of the ileum between two clamps. Left lower corner. Closure over a clamp of the distal end of ileum. Right lower corner. Clamp removed; the ends of the thread are pulled in opposite directions.

cecum and the latter with the ascending colon are separated from the posterior abdominal wall and turned medialward; the peritoneal fold just above the hepatic flexure is cut and farther medialward the gastrocolic ligament is divided so as to mobilize the lateral third of the trans-

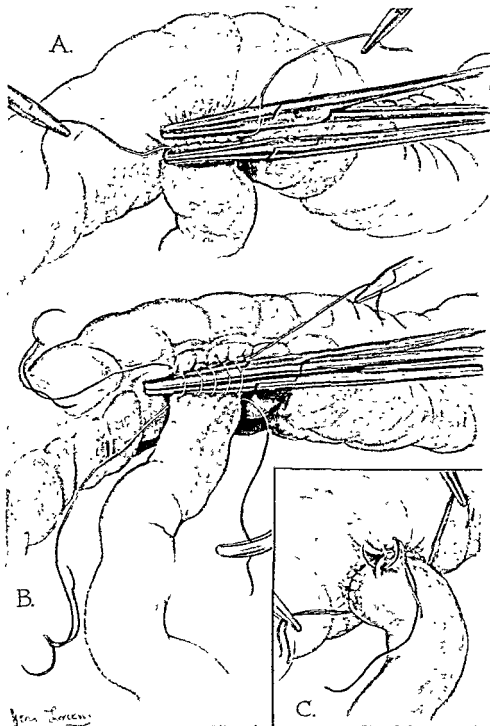


FIG. 229. Right hemicolectomy. End-to-side ileocolostomy. A. Seromuscular suture of the posterior lips. B. Seromuscular suture of the anterior lips. C. The clamps removed; the corresponding ends of each thread are tied to each other. Placing a reinforcing layer of sutures.

verse colon. The hepatic flexure with the lateral third of the transverse colon are then separated from the second and third portions of the duo-

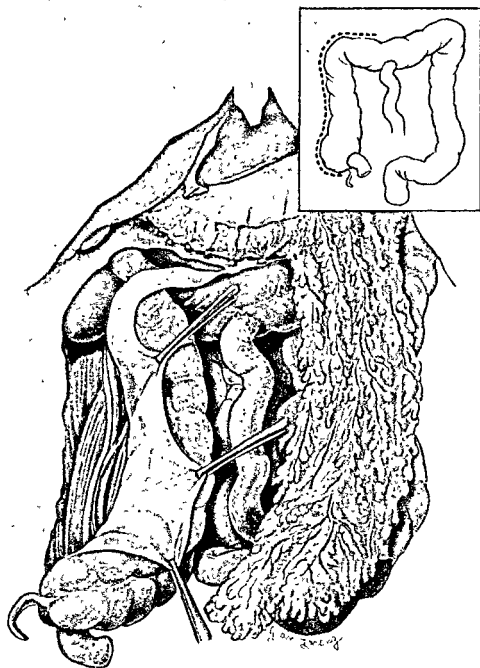


FIG. 230. Right hemicolectomy. Mobilization of the cecum, ascending colon and hepatic flexure.

denum and from the greater curvature of the antrum pyloricum, so that the cecum and ascending colon hang on their mesentery. The fat and lymphatics are stripped off the posterior abdominal wall while lifting

the mesocolon. However, one has to remember that while removing the lymphatics one should not lift the right ureter and the right spermatic (or ovarian) vessels which then will be found attached to the mesentery.



FIG. 231. Right hemicolectomy. The cecum, ascending colon and hepatic flexure have been removed. Peritonization of the right colon bed.

If that took place, they have to be stripped off the mesentery and dropped back on the posterior abdominal wall. Two crushing clamps are now placed on the transverse colon in a moderately oblique manner at a distance of one inch from the ileo-colic anastomosis; the bowel is

divided between the clamps by cautery; the mesentery is clamped by forceps and the bowel removed. The open end of the remaining portion of the transverse colon is closed over the clamp by the Parker-Kerr method.

Next, the edge of the cut mesentery is sutured to the lateral lip of the parietal peritoneum thus covering what formerly was the bed of the cecum and ascending colon.

In a two-stage resection the first consists of dividing the terminal ileum, closure of the end of the ileum attached to the cecum, and ileocolostomy and the second stage is carried out five to seven days later which consists of mobilization and excision of the bowel in the manner described above.

B. RESECTION OF THE SPLENIC FLEXURE. The technic follows the general pattern of the removal of the hepatic flexure just described. It is technically more difficult than resection of the hepatic flexure on account of its higher position. The technic is as follows:

Step 1. The abdominal incision is carried parallel to and immediately below the left costal arch, starting at the ensiform process and reaching the lowermost portion of the arch. In cutting through the soft tissues one will divide the left m. rectus abdominis in the medial part of the incision and the oblique muscles in the lateral part.

The mobilization of the bowel is started by cutting the left half of the gastrocolic ligament and gradually reaching the highest point of the splenic flexure. The advantage of starting from the stomach is that one can pull on that viscus (moderately, so as not to produce shock), instead of the bowel which is liable to tear.

After the left side of the transverse colon is sufficiently freed, a small incision is made through the parietal peritoneum just lateral to the upper portion of the descending colon; the index finger of the left hand is insinuated and the peritoneum is separated from the underlying tissues in an upward direction up to the summit of the splenic flexure and downward to the limit of the intended section of the bowel and cut. The bowel is now retracted medialward with the fat and lymph-tissue lying on the posterior abdominal wall. The bowel thus is mobilized on each side of the summit of the splenic flexure, which can now either be brought down and the holding band cut under direct vision, or the finger can be passed over the summit of the splenic flexure and the band cut over the finger so as not to injure the bowel. The bowel is now brought outside. One should carefully palpate the mesentery of the descending colon, as the left ureter and the ovarian (resp. internal spermatic) vessels may be closely attached to the mesentery. If they are, they should be stripped off the mesocolon and dropped back. The bowel is now resected well beyond the affected part and the continuity of the tract reestablished.

lished by end-to-end anastomosis either by the open or the aseptic over clamp method. In order to facilitate the work on the colon, a preliminary cecostomy is advisable; if that has been done, the cecostomy opening is still retained at least for 2 weeks after operation to serve as a safety valve by preventing increased pressure upon the suture line.

C. RESECTION OF THE DESCENDING COLON AND RECTO-SIGMOID. If the sigmoid is involved, it is removed well beyond the affected tissue, and the reestablishment of continuity is carried out at the time of removal of the diseased gut either by a primary suture method or by an aseptic over clamp method as described above, or else as a many-stage operation.

Up to recent times it was considered a dangerous operation, the one stage resection by the open method, in which the continuity of the intestinal tract was reestablished by open suture method, on account of high mortality rate due to peritonitis. Only after the introduction of sulfasuxidine, which nearly eliminates the *B. coli* from the intestinal flora, the open method became much safer and is gaining more and more advocates on account of its obvious advantages over multiple stage operations. In case of multiple stage operation it may be done either by Mikulicz exteriorization or by the "Obstructive resection method" of Rankin.

MIKULICZ EXTERIORIZATION TECHNIC. This is a four stage operation.

First stage. The abdomen is opened by a left paralateral rectus incision. The sigmoid is located. The adjoining portion of the descending colon is mobilized and turned inward together with the sigmoid. The mesentery of the sigmoid is ligated and cut in a "V" shape (it should be kept in mind that not only the blood vessel distribution but also the area of lymphatic drainage should be considered before deciding the amount of bowel to be removed). The portions of the bowel lying in close proximity to the part to be removed are laid side by side. A seromuscular suture connects them. Next, a suture fixes each loop of the "double-barrel" to the peritoneum, so that the diseased portion to be removed lies upon the skin.

Second stage. This is done four to seven days after the first stage. It consists of cutting off the bowel lying on the skin, leaving only one to one and one-half inches of the bowel above the skin level.

Third stage. This is carried out seven days to two weeks after the second stage and consists of introducing the blades of an enterotribe or of any strong artery forceps into the lumen of each opened bowel. The lock of the artery forceps is closed tightly. Each day the artery forceps is locked one notch tighter than on the previous day. In a few days the intestinal wall between the two loops is crushed, so that a spur no longer remains.

Fourth stage. Two to three weeks after the third stage the lumen of

the bowel is closed. The technic of closure is as follows: a circular skin incision is made around the colostomy opening. The incision is carried to the fascia in fat patients and through it and the muscle down to the peritoneum in lean patients. The intestinal tube is separated from the skin (in fat patients) and also from the fascia and muscle in lean patients. The opening of the bowel is now closed by inverting the edges by a Connell suture.

A second layer of seromuscular sutures is placed over the first layer. The suture material used is fine intestinal chromic catgut.

The skin (in fat patients), or muscle, fascia and skin in lean patients are closed over the sutured bowel. One has to be sure, that before starting to suture the bowel lips, there should be left no indurated pieces of fat attached to the edges of the gut. If pieces of indurated fat or of appendices epiploicae are attached to the intestinal wall, inversion of the edges is done under tension and as soon as the catgut is absorbed, eversion and pouting of the intestinal mucosa takes place and results in an intestinal fistula.

"OBSTRUCTIVE RESECTION" (RANKIN) METHOD. This operation is performed in two or in three stages. The first consists of removal of bowel harboring the malignant growth; the second of removal of the colostomy spur produced in the first stage of the operation; and the third of closure of the colostomy opening.

Technic: First stage. The abdomen is opened. The involved bowel is exposed; if it is the transverse colon, it is detached from the omentum; if it is the sigmoid, it is mobilized and brought outside the abdominal cavity. The blood vessels supplying the bowel to be removed are ligated close to the root of the mesentery and the intervening mesentery with its lymphatics are removed. Rankin's three-bladed clamp is applied to the two limbs of bowel just proximal and distal to the bowel to be removed. The bowel is cut away flush from the blades with a cautery. The mesentery is closed with exception of a small place through which the lips of the abdominal wound are sutured each to other. However, before suturing the lips of the abdomen the raw surface of the posterior abdominal wall produced by mobilization of the colon is peritonized. The abdominal wall is closed above and below the protruded ends of bowel.

The bowel is left thus obstructed for 48 to 72 hours. If the patient has gas pains the clamp on the proximal loop is released for a few minutes.

The *second stage* consists of the application of the enterotribe to crush the spur. This can be done either with a heavy enterotribe or any Ochsner or Kelly-Péan forceps. Rankin advises to destroy the spur 6 weeks after the 1st stage.

The *third stage* consists of closure of the colostomy opening. This is done in the manner described under "Mikulicz Operation."

EXCISION OF RECTUM (Figs. 232, 233, 234, 235). Of all the methods of excision of the rectum the radical abdominoperineal operation, as described and practiced by Miles, gained universal recognition.

The resection can be done as a one or a two stage operation. Miles' operation is a one stage operation. The first part of this one stage operation is the abdominal and the concluding part the perineal approach.

The patient is placed in the pronounced Trendelenburg position. A right paramedian incision, half an inch away from the midline is made, starting one inch above the umbilicus and reaching the crest of the pubic bone. After the abdomen is opened a self-retaining retractor is placed. Next, the abdomen is carefully examined for metastases and for this purpose the pelvic mesocolon should be examined first for nodules. They may be found along the line of attachment of the mesocolon to the posterior abdominal wall, or along the course of the inferior mesenteric and superior hemorrhoidal vessels; or on the mesentery along the border of its attachment to the colon; or in the substance of the mesentery itself. If nodules are found close to the posterior abdominal wall, this signifies that wide extramural extension of the disease already has taken place and the case is inoperable, because recurrence is almost certain to occur. Next, the liver is examined. One should then examine the urinary bladder and the vagina. If the bladder or the vagina are involved the case is inoperable. If exploration shows that the case is operable, the following steps are made:

Step 1. The small bowels are pushed away from the pelvic cavity upward and the pelvic cavity and the immediate vicinity of the lower lumbar region is walled off from the rest of the peritoneal cavity.

Step 2. The pelvic colon is brought outside the abdominal cavity. If it is difficult to do so on account of shortness of its mesentery, the sigmoid can be mobilized by cutting the parietal peritoneum lateral to it.

Step 3. The inferior mesenteric vessels are ligated at a point between the origin of the first and second sigmoid arteries. This point is about at the level of the bifurcation of the abdominal aorta. This can best be accomplished by passing an aneurysm needle around the vessels, grasping with them some of the mesocolon tissue. One has to be careful not to include in the ligature the left ureter, which is situated at about three-fourths of an inch to the left from the inferior mesenteric artery at the level of the bifurcation of the aorta, but at the level of the promontorium they lie close to each other, for which reason ligation of the inferior mesenteric artery is much safer at the level of the bifurcation of the aorta. Another ligature is placed about one half of one inch below the first ligature and the vessels are divided between these two ligatures. This renders the operative field in the subsequent steps nearly bloodless.

Step 4. The parietal peritoneum is cut at right side of the mesocolon parallel to the bowel starting immediately below the upper ligature

placed on the inferior mesenteric artery and cutting it as far downward as the promontorium. The finger is introduced behind the bowel and places on the stretch the peritoneum to the left of the rectum; it is then cut immediately to the left of the rectum, thus avoiding injury to the left ureter.

Step 5. The finger introduced behind the rectum separates it from the

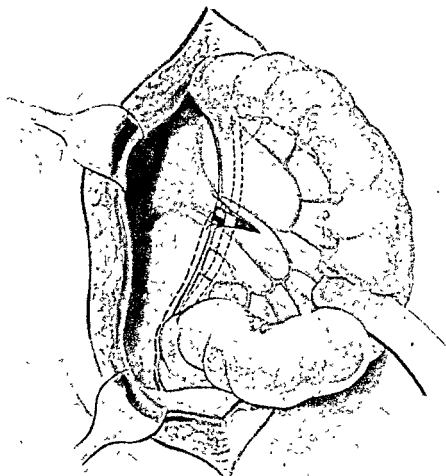


FIG. 232. Abdomino-perineal resection. Ligation of the inferior mesenteric artery.

bone all the way downward as far as the sacrococcygeal articulation; and while elevating the bowel from the sacrum the finger raises also the pelvic peritoneum, which is divided on either side parallel and close to the brim of the true pelvis as far as the base of the bladder, care being taken not to injure the ureters. Then these incisions are turned medially until they meet each other behind the base of the bladder in the male or the upper part of the vagina in the female.

Step 6. The anterior wall of the rectum is separated from the vesicula

seminalis and the base of the bladder. This separation goes downward as far as the base of the prostate.

Step 7. After the rectum has been freed posteriorly to the sacrococcyg-

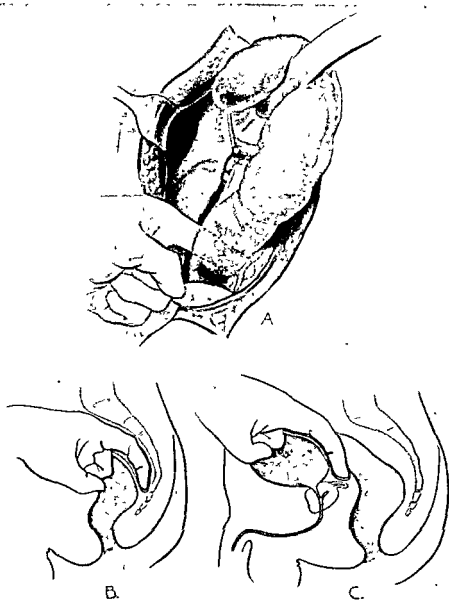


FIG. 233 Abdomino-perineal resection. A. Mobilization of rectum. B. Separation of rectum from sacrum. C. Separation of rectum from bladder.

cal joint and anteriorly to the base of the prostate, the lateral attachments of the rectum can be readily felt. They consist on each side of a broad dense band of connective tissue which passes from the lateral wall

of the rectum downward and lateralward toward the base of the bladder. The middle hemorrhoidal artery passes through each ligament and, therefore, these ligaments have to be cut between clamps.

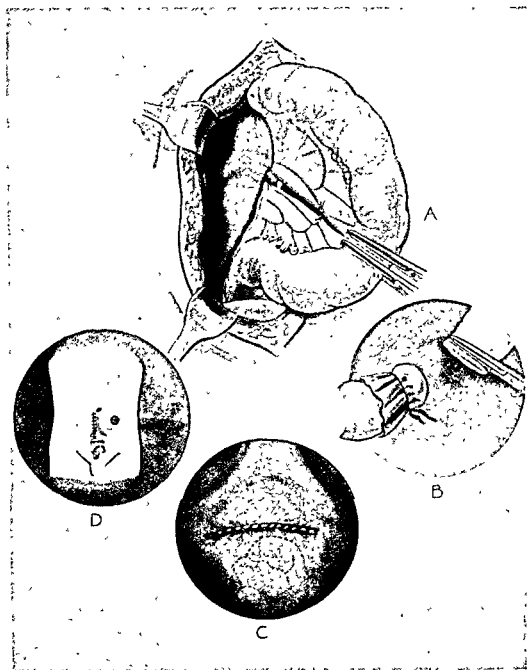


FIG. 234. Abdomino-perineal resection. A. Cutting across the sigmoid loop. B. Tying of the distal sigmoid loop over a rubber dam. C. Suture of the vesical peritoneum to the promontory. D. Final view after completion of the abdominal stage of resection.

Step 8. The pelvic colon is divided between 2 clamps at a point 3 inches below the descending colon. Each end is covered with a piece of rubber and tied. The occluded distal end of the colon is pushed into the pelvic

floor as far down as possible with the tied end lying at the lowermost position, so that it can easily be reached through the perineum.

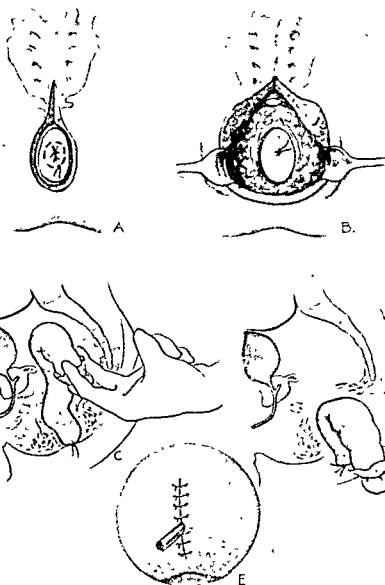


FIG. 235. Abdomino-perineal resection. A. Closure of anus by a purse-string. Cutting of the skin around the anus. B. Exposure of gluteus and levator ani muscles. C. The distal end of the distal loop of sigmoid is brought outside. D. The distal loop is already cut away from the surrounding structures. E. Final view of the perineal stage of resection (diagrammatic).

Step 9. A new pelvic floor is now formed above the true pelvis by suturing the peritoneum attached to the bladder to the peritoneum in front and above the promontory.

In women the broad ligaments instead of the vesical peritoneum can be utilized for this purpose.

Step 10. The proximal end of the bowel is brought outside through a small opening lying on the spino-umbilical line at a distance of $1\frac{1}{2}$ inches from the spina iliaca anterior superior or through the same opening through which the operation has been performed.

PERINEAL STAGE OF THE OPERATION. The patient is placed on his abdomen so that the lower end of the body is at the edge of the table and the legs hang down at their entire length, or he may lie on the table with the pelvis elevated.

Step 1. The anus is closed by a purse-string suture. A longitudinal incision starting at the sacrococcygeal joint is running in the internatal furrow down to one inch behind the anus and then to the right and left of the anus, and thence down in the form of a horse-shoe, after which the anterior ends are joined by a transverse incision in front of the anus. It is important that a wide area of perineal skin should be removed, because the skin in this area is particularly prone to manifest recurrences. This means that the arms of the above outlined horse-shoe should be wide.

The gluteal flaps are reflected and retracted, thus laying bare the coccyx.

Step 2. The coccyx is removed.

Step 3. A small transverse incision immediately below the sacrum is made and the fascia propria recti is detached from the ventral surface of the lowermost portion of the sacrum. From there the index finger goes upward in front of the sacrum and enters in that part of the abdominal cavity which lies below the pelvic floor formed in the abdominal stage of the abdominoperineal resection. A transverse incision is made through each coccygeus muscle extending from each side of the rectum laterally to the great sacrosciatic ligament.

The bowel is now caught at its end covered with rubber and drawn outside. Traction is now made on the bowel and the *m. levator ani* of each side comes into view. The levators are now divided at their origin from the lateral pelvic wall.

The anterior wall of the anal canal is detached from the central point of the perineum, care being taken not to injure the membranous portion of the urethra. The large cavity thus produced is now packed with gauze.

The skin from below is closed, leaving only a small opening for the end of the gauze drain.

Diverticulitis

Diverticulitis of the colon in many instances is treated medically; some cases belong to the domain of elective surgery; and only seven to eight per cent of them become a subject of urgent surgical intervention

on account of complications, such as perforation, abscess formation and acute intestinal obstruction.

ETIOLOGY. A diverticulum is a sacculation projecting from different portions of the digestive tract. In the intestine it may spring from the mesenteric, antimesenteric and lateral intestinal walls. Most frequently it occurs in the sigmoid. As a rule, it is multiple. They are of two varieties—congenital (true) and acquired (false), the latter being preponderant. Diverticula usually occur in patients past middle age, being rare under the age of thirty, uncommon under the age of forty and usually are encountered between the age of forty and fifty. Nearly 5 per cent of the patients coming to autopsy show diverticula. It is more frequent in men than in women, the ratio being 3:2. The diverticula become inflamed in about 30 per cent, 25 per cent of which require surgery. In other words, only 7 to 8 per cent of patients with diverticulosis develop diverticulitis demanding operative intervention. In about 80 per cent the diverticula are located in the sigmoid, the remaining 20 per cent being situated in the descending colon, rectum, transverse colon and ascending colon. A diverticulum may be present even in the appendix.

PATHOLOGY. The diverticula are bottle-shaped with a narrow neck. As long as the intestinal contents freely enter and leave the sac, no inflammation takes place. If, however, the drainage from the diverticulum is impeded, stagnation and infection with ensuing inflammation take place. This inflammation may be confined to the sac itself, or spread to the surrounding tissues, producing peridiverticulitis. Inflammation may affect the mesentery resulting in its thickening. The bowel may become edematous, the surrounding inflamed tissues may compress the bowel, thereby producing intestinal obstruction. The thickened mesentery and peridiverticulitis produce tumefaction which may be mistaken for malignancy. The process may repeatedly subside and become exacerbated, thus producing chronic inflammation. From time to time the inflamed mass may break into another bowel loop, the urinary bladder or outside through the skin with resulting fistulae.

SYMPTOMATOLOGY. The symptoms vary, depending on the stage of inflammation, the segment of the bowel involved, the presence or absence of complications and the type of complications.

If the inflammation affects a diverticulum of the cecum, the symptoms may simulate acute appendicitis, such as pain, tenderness, rigidity and leukocytosis. In the presence of abscess formation it may be impossible to differentiate it from an appendicular abscess.

If the process takes place in the sigmoid, the symptoms are the same as on the right side with this difference only that the pain, tenderness and rigidity are localized in the left lower quadrant. One may then suspect acute diverticulitis, particularly in obese, middle aged persons. The symptoms will also vary depending on the complications. If the diver-

ticulum perforates into the free peritoneal cavity, general peritonitis will ensue with its characteristic manifestations; if the diverticulum perforates into a part of the peritoneal cavity walled off by adhesions, localized abscess formation takes place. If the diverticulum breaks into the urinary bladder, the urine will acquire a fecal odor, bubbles of gas and even particles of feces will come away with the urine. In case the diverticulum breaks into another intestinal loop, clinical remission or even recovery takes place.

In chronic diverticulitis the patient has abdominal discomfort, change of bowel habit, from time to time local tenderness and rigidity. In many cases the symptoms subside entirely only to reappear at some future time.

DIAGNOSIS. Acute cases present great diagnostic difficulties as is attested by the fact that they have been mistaken for appendicitis, cholecystitis, pancreatitis, pelvic tumors, carcinoma and intussusception.

In chronic cases the diagnosis is simple, as it is nearly always cleared up by a radiogram.

TREATMENT. If no complications arise this disease is treated medically. It consists of putting the patient on a low-residue diet and the administration of mineral oil in order to prevent complications. If these do arise, they have to be treated surgically.

Perforation is treated by closure with inversion of the edges. If, however, the diverticulum is indurated and is impossible to close the opening, the bowel is exteriorized, the opening left intact with a view to future closure or resection of the affected segment of the bowel.

Abscesses have to be incised and drained.

Spreading peritonitis is treated in the usual manner (penicillin, sulfa drugs, suction tube, intravenous infusions, blood transfusions or plasma infusions).

Chronic diverticulitis is treated surgically only when complications arise.

Intestinal obstruction is treated by preliminary decompression followed by the excision of the part involved. If the latter is inaccessible in the pelvic cavity, or reveals extensive adhesions, permanent colostomy may be required.

Fistulae are treated by their dissection, and closure of each end. In some cases it is a comparatively simple procedure; in others it may prove a difficult surgical intervention, as, for instance, in a sigmoidovesical fistula in which the tract connects the sigmoid with the base of the bladder.

PROGNOSIS, regardless of the type of treatment (medical or surgical) is not good. The mortality in medically treated cases is about 30 per cent and in the surgical cases it varies greatly from 10 to 60 per cent.

Traumatic Injuries of the Rectum

Injuries of the anus and rectum date back to remote antiquity. The Chinese have put bamboo sticks into the rectum as a punishment for adultery. The Code of Hammurabi prescribed impalement by "sitting on a stick" as a punishment for lustful murder. As late as in the sixteenth and seventeenth centuries, Cossacks punished offenders by "sitting on a stick" or by breaking them on the wheel or quartering.

ETIOLOGY. Injury of the rectum may be produced by gunshot, stabbing, impalement with a pitchfork, sigmoidoscopy, the improper introduction of an enema nozzle and even by sexual perverse acts.

PATHOLOGY. Rectal lesions are either extraperitoneal, if located below the peritoneal reflection, or intraperitoneal. There may be a single lesion or also involvement of the urinary bladder, small intestine, pelvic bones or the large pelvic vessels. While improper therapeutic or diagnostic instrumentation usually produces a single lesion, traumatic accidents often result in multiple, complicated injuries.

SYMPTOMATOLOGY. The symptoms vary according to the nature and extent of the lesion or lesions, with particular reference to the presence or absence of involvement of the peritoneum and the length of time that has elapsed since receipt of an injury.

A single intraperitoneal injury seen early manifests only moderate bleeding from the rectum. The patients ordinarily complain of only slight pain. But a few hours after receipt of such an injury there is marked distention of the abdomen, pneumoperitoneum, obliteration of the hepatic dullness and tenderness and rigidity of the lower abdomen.

Lesions below the peritoneal reflection may cause no more than slight bleeding through the rectum; if retroperitoneal infection sets in the symptoms may be those of sepsis.

Involvement of the bladder yields the typical symptoms due to cessation of spontaneous micturition. The urine may escape through the rectum and blood may be forced out through the urethra. One often notes dullness in the suprapubic region, tenderness and rigidity of the lower abdomen, and a frequent desire to micturate.

DIAGNOSIS. A correct diagnosis is arrived at partly by the history of a preceding trauma and partly by physical examination. Digital exploration alone may reveal a perforation of the rectosigmoid extending to the peritoneal cavity, but proctoscopy or sigmoidoscopy may have to be added to clinch the diagnosis. It should, however, be borne in mind that a small perforation of the rectum has been overlooked and found only at autopsy.

TREATMENT. A single, extraperitoneal lesion may require no more than the insertion of a large rubber tube into the rectum with or without its fixation to the anus. An intraperitoneal injury requires determination

whether it is isolated or complicated. This is a problem which is not easily solved in the early stage. While the situation becomes clear if the bladder is involved, a complicating lesion or lesions of the small intestine may at first yield no symptoms. To remain expectant until signs and symptoms appear, spells doom for the patient. For this reason every injury of the rectum that extends to and into the peritoneal cavity demands immediate exploratory laparotomy, which may be carried out abdominally, through the perineum or transsacally. Exploration through the abdomen is undoubtedly the best procedure because it affords wide exposure and a good survey for reparative action.

If it is found that the injury is limited to the rectum and involves a comparatively small area, it may suffice to close the opening in two layers, dust the wound with sulfanilamide and close the abdomen in the usual manner without drainage. A large rubber tube is introduced into the rectum and allowed to protrude through the anus. Should the opening of the rectum be too large for simple closure, omentum must be drawn upon for reenforcement, a drain carried outside the abdominal cavity and a large rubber tube introduced through the rectum.

If the injury also involves the bladder, the opening in it is closed as well as that in the rectum. An indwelling catheter is left in the bladder and a large rectal tube is carried through the anus. In addition, a drain is placed between the rectum and the bladder and brought outside through the abdominal wall.

An extraperitoneal injury to both the bladder and the rectum ascertained by laparotomy calls for closure of the peritoneal cavity and a suprapubic approach through the space of Retzius. After closure of the openings in the bladder and the rectum an indwelling catheter should be left in the former and a large rubber tube in the latter led through the anus. Drainage of the space of Retzius completes the operation.

The mortality from complicated injuries in the period preceding the introduction of plasma and chemotherapy has been appallingly high, reaching as high as 60 per cent. The present effective measures available to combat shock and infection have considerably lowered that rate.

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Chapter XVII

Perforative Peritonitis

By ELPIDIO STINCER

A clinical study of peritonitis due to perforation should be preceded by an anatomic review, and also by a brief excursion into the domain of its physio-pathology, because on them rests the diagnosis and surgical treatment of this interesting group of acute abdominal lesions. In fact, peritonitis due to perforation of the appendix is not the same as peritonitis due to gastro-duodenal perforation neither in their clinical manifestations nor in their course. Likewise, it is not the same in its course nor in its prognosis in case of acute gastric perforation through the anterior gastric wall; also we cannot consider of equal gravity peritonitis of the upper portion of the abdomen and that of the lower portion. The anatomic conditions vary and for this reason also the clinical manifestations and the prognosis. It is a well known fact that the greater danger is associated with lesions of the upper portion of the abdominal cavity, due to the proximity of the diaphragm, where there exists a great richness of the lymphatics and whose relations to the heart, pleura and lungs explain the greater danger of peritonitis in the supramesocolic region.

From the foregoing, therefore, it can be deduced that in order to comprehend the evaluation of clinical symptoms of perforative peritonitis it will be necessary to keep in mind the anatomic position and the relationship of the perforated viscus to other structures; the detailed structure of the viscus itself; the site of perforation; its cause; the causative germs and the degree of their virulence; the circumstances in which perforation took place; the state of the patient, and the like.

All these factors will help us to explain the surgical considerations which govern the diagnosis, prognosis and treatment of perforative peritonitis.

We shall divide our exposition into two large sections: (a) Anatomic and physiopathologic considerations; and (b) Clinical study.

Anatomic and Physiopathologic Study of Peritonitis Due to Perforation

This section we shall subdivide into: 1. Study of the peritoneum; and 2. Study of perforations *per se*.

Study of the Peritoneum

The peritoneal serosa is the structure that puts an especial mark on a great group of abdominal affections.

The peritoneum covers without interruption the great abdominopelvic cavity, thus constituting its natural borders. But whereas in a morphologic sense we cannot separate the abdominal from the pelvic peritoneum, we will nevertheless, from the medico-surgical aspect and even for the sake of clearness of presentation, accept such a division.

ABDOMINAL PERITONEUM. The abdominal peritoneum enters into intimate relationship with different viscera and, therefore, shows various arrangements. From it are formed different folds under the names of omenta, mesenteries and ligaments, which form connections between the viscera, between the viscera and parietes, be these viscera strictly of the alimentary tract or not.

As all structures of serosal type, the peritoneum consists of two layers: a superficial, which is endothelial, and a deep layer of connective tissue. At the same time, it is described as a parietal and a visceral layer and between them is situated the peritoneal cavity. The former layer covers without interruption the entire surface of the abdominal wall, the latter is related to various viscera, forming more or less complete envelopes.

In order to study the visceral peritoneum of an adult, it is necessary to know about the visceral peritoneum of an embryo, because it is known that in the course of the development the abdominal portion of the digestive tube is found to consist of only 4 loops; these are fixed to the posterior abdominal wall by a continuous dorsal mesentery, which can also be subdivided into four ligaments or mesenteries—one for each loop. The four primary loops are from above below, the gastric loop, which gives origin to the abdominal portion of the esophagus, to the stomach and to the first portion of the duodenum; the duodenal loop, which gives origin to the remaining three portions of the duodenum; the umbilical or vitelline loop that gives origin to the jejunum, ileum, cecum,

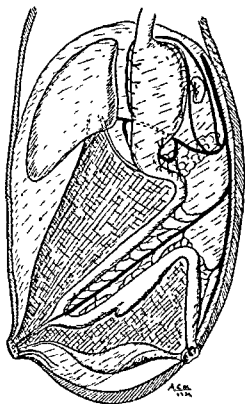


FIG. 236. The primitive state of the peritoneum and digestive tube of an embryo.

colon ascendens, colon transversus down to the splenic flexure; and, finally, the terminal loop, which gives origin to the colon descendens, ileo-pelvic colon and the rectum. At the apex of the vitelline loop is implanted the vitelline duct. To each of these loops belongs a segment of the continuous dorsal mesentery; therefore, there are a posterior mesogastrium, mesoduodenum, umbilical or vitelline mesentery and a terminal mesentery. In an adult, due to certain changes and modifications that take place in the digestive loops, whether due to torsion or to vascular arrangements, these mesenteries also undergo changes, with the exception of the mesoduodenum, which remains totally adherent or fused. The others usually consist of two parts: one, which remains free, nonfused; the other, which fuses or adheres either directly to the parietal peritoneum or to other viscera, thus forming fused zones of the peritoneum or fused fascia. Knowledge of these fused zones is absolutely necessary for the surgeon in order correctly to mobilize some of the abdominal structures which lie deep and are fixed, such as the duodenum, pancreas and so on, and of which modern surgery took advantage, thus enabling their introduction into its domain.

The same digestive tube is found fixed, but on this occasion only partially, to the anterior abdominal wall. Therefore, here exists an anterior ventral peritoneal fold, which is suspended and interrupted in its center. The lower portion is confined to the preanal portion of the bowel, from which take origin the urinary bladder and the urachus; the upper portion is marked by a reticular recess, which will form the liver.

For what was expounded it is clear how variable will be the disposition of the visceral peritoneum in an adult in relation to numerous changes pertaining to the direction of different digestive organs, as well as to the development of different fused fascia and again to different accidents in

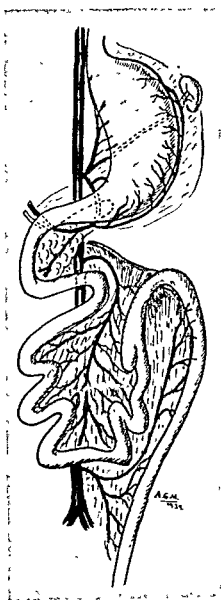


FIG. 237. The first stage of rotation of the digestive tube.

development of different viscera or of the peritoneal fold (Figs. 236, 237, 238).

PERITONEAL CAVITY. We already have seen the manner of formation of the peritoneal cavity; we know the complications that this large

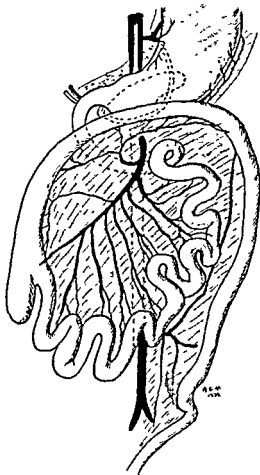


FIG. 238. The last stage of rotation of the digestive tube.

serosal cavity offers us and this is essentially due to the irregularity of the arrangement of the visceral peritoneum; to numerous folds (omenta, mesenteries, ligaments) which form numerous compartments thus making it, at least theoretically, indescribable. This anatomic subdivision into compartments is considerably increased during the development of pathologic processes by virtue of one of the best properties of the serosa, namely, the property to form adhesions, thus regaining its primitive properties during its embryonic stage. In this manner is achieved the process of spontaneous recovery by localization of different morbid processes and giving a place, as we will see it later, to a clinical group of localized or encysted peritonitis with a relatively favorable prognosis which has to be differentiated from general or

diffused peritonitis with a particularly grave prognosis.

PERITONEAL FOLDS PRODUCING COMPARTMENTS IN THE GREAT-PERITONEAL CAVITY. These, as was stated, consist of mesenteries, omenta and ligaments (Figs. 239, 240).

Mesenteries. When we speak of the embryonic stage of the digestive tube and its folds (anterior or ventral and posterior or dorsal), we imply that due to certain changes and modifications in the evolution of the same, some anatomic changes take place producing on one hand fused

fascia and on the other hand some free, mobile parts, which, resembling an embryonic type, become differentiated as the transverse mesocolon, which is nothing else but a portion of the non-fused mesentery of the

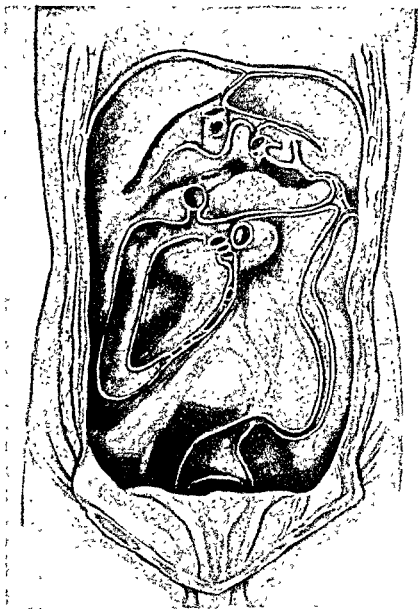


FIG. 239. The viscera are removed; note the compartments in the peritoneal cavity formed by peritoneal folds.

primitive umbilical loop; another part forms the mesentery of an adult, which represents the other portion of the same fold neither of which fuses. I wish to say then that the mesentery of the primitive vitelline loop consists in an adult of three portions completely differentiated: one is fixed, adherent, fused to the peritoneum of the posterior ab-

dominal wall and which is known as the right colo-mesocolic fascia; the other consists of two free, non-adherent, non-fused portions one of which constitutes the mesentery of an adult, and the other the transverse mesocolon.

The transverse mesocolon is a peritoneal fold which forms the great dividing partition of the abdominal cavity. It is this fold that separates,

though incompletely, the superior portion of the abdominal cavity from the lower one. It is also the transverse mesocolon which establishes the great anatomic division of peritonitis as supra- and infra-mesocolic.

The mesentery of the jejunum and ileum is another partition equal to the one we have just described and divides, also incompletely, the lower abdomen. Indeed, the mesentery, extending from the left of the second lumbar vertebra to the right sacro-iliac articulation up to the ileo-cecal angle follows a more or less oblique course from above below and from left to right and divides the lower abdomen into two large segments—the right and left.

It suffices to grasp the transverse colon with its mesentery and pull them outside to render them tense and on the other hand to grasp the jejunum and ileum with their mesenteries and also draw them outside

clearly to demonstrate the division of the great abdominal cavity into two compartments: the superior or supramesocolic and the inferior or inframesocolic. The latter is again subdivided into two parts—one, situated above and to the right of the mesenteric partition which we call the right superior recess, and the other corresponding to the part inferior and left from the mentioned partition which we call the left inferior recess. Here are two compartments where circumscribed peritonitis may be established and in which natural drainage by gravity cannot be equally well established, because in the right, upper recess there is difficulty for evacuation on account of an impediment offered by the mesentery, so that it can gravitate only into the right iliac fossa and remain there; therefore, if it takes origin there, it will remain there or

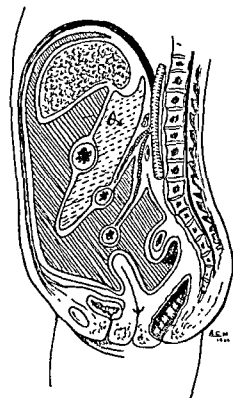


FIG. 240. Sagittal section of the peritoneal cavity. The Foramen of Winslow (see arrow) is the beginning of the omental cavity.

spread above and beyond this fossa (peritonitis of appendicular origin, appendicular abscess); while in processes developed in the left inferior recess the contents easily drain into the pelvis and do not have a tendency to spread. Thus one can see that the gravity of peritonitis increases in proportion to our approach to thoraco-abdominal region (a region anatomically and clinically mixed; anatomically, because it contains abdominal and thoracic organs and clinically because peritoneal lesions here easily become complicated by cardio-pleuro-pulmonary processes).

Omenta. There are three omenta: the great or gastro-colic; the small or gastro-hepatic and the third—gastrosplenopancreatic.

The small omentum may be defined as gastric peritoneum stretching from the lesser curvature to the hilum of the liver. It divides the upper abdomen into three compartments: the right or the hepato-biliary; the left or gastrosplenic and the posterior which constitutes the posterior cavity of the omentum. The latter compartment forms a large peritoneal sac stretched from the front backward and extending in width from the foramen of Winslow to the hilum of the spleen and at the height from the lesser curvature of the stomach and upper border of the first portion of the duodenum to the lobe of Spigelius.

To this retroëpiploic cavity belongs the foramen of Winslow which constitutes the opening into this cavity and in which the presence of the hepatic pedicle (a. hepatica, ductus choledochus and vena porta) is sufficient to explain the gravity of pyloroduodenal lesions when they originate from the posterior surface of these structures. Next to the foramen is situated the vestibule of the cavity which communicates by medium of the foramen bursae omentalis with the retrogastric portion of the lesser peritoneal cavity.

The anatomic arrangement of this posterior compartment of the upper portion of the abdomen has an important relation to prognosis in ruptures of the posterior gastric wall, as well as in pathologic processes developing in this cavity, as they have a tendency to remain more or less silent, localized and protected, at least for some time.

The great omentum is nothing else but a part of the posterior mesogastrium secondarily fused with the transverse colon and its mesentery; it is a vast apron situated over the intestinal loops and beneath the parietal peritoneum.

The definition that we have given to the great omentum enables us clearly to understand the manner of breaking up the fusion which the surgeon utilizes as an excellent route of access to the omental cavity.

The gastrosplenopancreatic omentum is a union of gastrosplenic with pancreatospenic ligaments at the region of the hilum of the spleen. Therefore it is like an anatomic consolidation between these two omenta justifying the name of gastrosplenopancreatic omentum.

As far as the peritoneal ligaments are concerned it is worth mention-

ing the suspensory ligament of the liver and the cystoduodenocolic ligament. The suspensory ligament of the liver which forms an incomplete partition between the lobes of the liver—the right and the left—separates the abscesses of one from the other side, and, therefore, it is important to recognize its rôle as a barrier to transmigration of the hepatic accumulations from one lobe to another. As far as the cystoduodenocolic ligament is concerned, it is nothing else but a diverticulum of the small omentum, a prolongation of the same to the right. When it does exist, it does not produce pathologic processes *sui generis* unless when accidentally caused by inflammatory conditions at the zone which adjoins it, it becomes organized as fibrous cord which is capable of producing pathologic changes.

PELVIC PERITONEUM. In the pelvic peritoneum we also have parietal and visceral peritoneum, as well as folds built as ligaments or attachments. No omenta are present here.

The parietal peritoneum of the pelvis covers all the walls of the cavity and is creased by subperitoneal pelvic tissue.

In this subperitoneal pelvic space develop a series of anatomic structures which in their turn become isolated as chambers or compartments by fibrovascular layers of the pelvis. The visceral pelvic peritoneum in man differs from that of a woman and forms partitions of the pelvic cavity. In man the serosa covers the posterior surface and the middle of the superior parts of the lateral sides of the bladder. After that it reflects over the pelvic portion of the rectum forming the base of the vesicorectal pouch—a suitable diverticulum for damming peritoneal exudates.

In woman the uterus between the bladder and the rectum determines the formation of a base of the vesico-uterine sac in front and the recto-uterine sac of Douglas behind the uterus. On the other hand, from the posterior and inferior part of the uterus the peritoneum projects outside in the form of a fold to become attached to the sacrum as two semilunar folds—the folds of Douglas—which form the lateral boundaries of the base of the sac of the same name.

From the lateral sides of the uterus emerge the broad ligaments which attach this organ to the lateral walls of the pelvic cavity. As a result, the pelvic cavity in a woman is divided into two large compartments: anterior, vesico-uterine and posterior or recto-uterine; the Douglas folds subdivide the posterior into three compartments: a central or the base of Douglas' pouch and two lateral, right and left, in whose bases are seen shining the ureters and the internal iliac vessels.

It is not necessary to overemphasize the clinical importance of this topographic subdivision of the pelvis, because as a result of it there is a natural tendency of purulent accumulations to become localized or encysted, particularly because it concerns regions sloping from the general peritoneal cavity and in which the power of absorption is less, so that

these accumulations forge a passage in the direction of the vaginal or rectal cavities, thus producing spontaneous recovery of pelvic abscesses and phlegmons.

For the same reason colpotomy acquires a preponderant rôle in pelvic surgery.

The last peritoneal fold left for us to describe is the ileo-pelvic mesocolon. Its parietal attachment resembles the letter S and contributes to division of the pelvic cavity into two compartments: the superior right

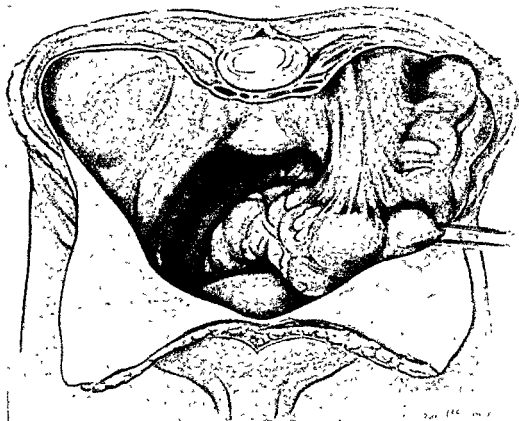


FIG. 241. Ileo-pelvic mesocolon; note the subdivision of the pelvis into superior and inferior compartments.

recess and inferior left recess, exactly in the same manner as the mesentery subdivides the abdominal cavity (Figs. 241, 242).

The physiopathology of the peritoneum we emphasized in the course of its anatomic description. This great serosal structure has definite defensive properties, especially when it is irritated or infected. Here are formed adhesions of different degrees ranging from simple agglutination with a possibility of complete absorption to permanent, very strong and resistant ones, as a result of peritoneal fibrosis. These adhesions, which in some cases have a definite defensive rôle, may in other cases become transformed into fibrous bands, render the viscera impossible of exer-

cising their physiologic excursions, leading to grave accidents as, for instance, intestinal obstruction.

The reaction of the peritoneum to the microbes varies depending on the character of the latter, their degree of virulence, as well as the local conditions and general state of the patient.

There are different ways by which microbes may affect the peritoneum—traumatic and non-traumatic. Generally, we may say that pathologic reactions following peritonitis depend on many factors.

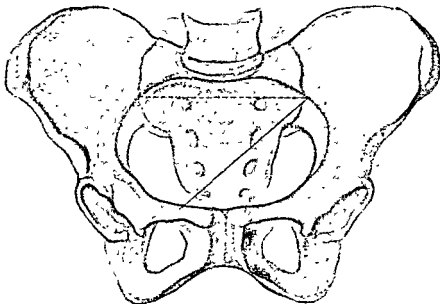


FIG. 242. Schematic presentation of the superior and inferior compartments of the pelvis.

The ability of absorption by the peritoneum increases in the acute and diminishes in the chronic processes. Reabsorption of inflammatory exudates is enhanced in the horizontal position and with movements of the bowel.

In peritonitis due to trauma we may say that the body and the peritoneum are in a better defensive position than when previous visceral lesions existed, such as typhoid, appendicitis, peptic ulcer, tuberculosis, neoplasm, lues, in which the local region is already infected, and which produce repercussions during general well being and decidedly increase the gravity of the prognosis.

Vascularization and innervation of the peritoneum are richer in the visceral region which explains their influence upon the general circulation and intestinal peristalsis in peritoneal inflammation. Paralytic ileus is due to paralysis of the nervous system of the intestine.

We could not close this section of the physiopathology of the peritoneum without referring to its most important formation, namely the great omentum. The main and incontestable function of the great omentum is plastic and phagocytic. Omentum is fundamentally a lymphoid organ of defense which has a tendency to attach itself to altered peritoneal points, be it perforation of a viscus, wound of the wall or of some inflammatory process. Omentum is utilized in surgery for peritoneal repair and many times its implantation into an opening produced by a rupture of a viscus may achieve a cure. In some cases, as in rupture of appendix, the omentum may spontaneously achieve in a manner more brilliant than that by the hand of the surgeon closure of the perforation, thus preventing diffusion of peritonitis with all its consequences.

Anatomic and Physiopathologic Study of Perforations

It seems logical that once having studied the peritoneum from a dual aspect, we should now consider its perforations, which will bring us to the clinical aspect of the problem. All abdominal organs may become the subject of the fatal accident of perforation (with its certain sequela of peritonitis), though not with the same frequency, such as the appendix, stomach, duodenum, jejunum-ileum, colon, rectum, gallbladder, urinary bladder, uterus, tubes, ovary, to which we may add rupture of some types of hepatic abscesses into the free peritoneal cavity.

Perforation is a grave abdominal condition which occurs relatively rarely in comparison with other acute abdominal conditions. Rupture may be due to traumatic and non-traumatic causes, but all will produce peritoneal syndromes which may differ in certain groups. For instance, a non-penetrating traumatic injury of the abdominal wall may occasionally produce a rupture of a hollow viscus, such as the stomach, intestine, urinary bladder, gallbladder, resulting in perforative peritonitis, the prognosis of which will depend on the injured viscus, the virulence of the contents that escaped through the rent, on the state of the viscus when rupture took place (full or empty) and on the time when surgical intervention took place.

As far as ruptures of non-traumatic origin are concerned, we mention ulcers of peptic origin (gastro-duodenal, Meckel's diverticulum) and those produced by typhoid fever, neoplasms and by specific infections (tuberculous, luetic). Occasionally we encounter perforations which are neither of peptic origin nor specific in character (simple ulcers of the small bowel) without being able to determine their pathogenesis even after histopathologic examination.

GASTRODUODENAL PERFORATIONS. Here the most frequent type is perforation of ulcerous origin, which may take place either during the course of an acute or a chronic ulcer. Gastric perforation presupposes dissolution of the continuity of all layers of the gastric wall from the mucosa to the serosa; this may take place at once in a violent manner or slowly by successive steps. Whatever the mode of perforation, it forms a pathologic complex of an infectious type (Duval) combined with mechanical factors.

The *site of perforation* does not run parallel to that of the ulceration because ulcers that most frequently perforate are on the anterior or posterior wall of the stomach in the vicinity of the pylorus, which in the

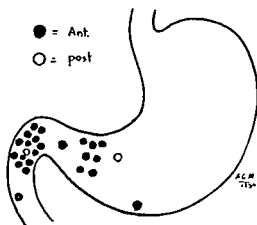


FIG 243. The common locations of gastro-duodenal perforations. Filled circles indicate sites on anterior wall, empty circles those of posterior wall.

majority of cases opens into the free peritoneal cavity. The ulcers on the lesser curvature as well as those of the upper and lower borders of the pylorus rupture less frequently owing to the presence of omenta at the borders of the stomach, whose defensive rôle we already emphasized. Therefore, we can anatomically divide the gastroduodenal perforations into perforations of the poorly protected wall with a bad prognosis, and perforations at the curvatures close to omenta, as better defended and of a less grave prognosis (Fig. 243).

Rupture of the posterior gastric wall faces the omental cavity for which reason it is less serious than rupture of the anterior wall, because in the former peritonitis does not spread early on account of the natural tendency of localization of the effusion. For this reason this type of posterior perforation gives a more or less obscure symptomatology, a silent one, which surgeons should keep in mind. Perforation of the posterior surface of the pylorus due to close relation of this organ to the hepatic pedicle is likely to aggravate the prognosis of peritonitis.

As far as perforations along the curvatures are concerned we can say

that though the prognosis is less grave on account of the proximity of the omenta, they may nevertheless produce grave hemorrhages and damage to the extrinsic nerves of the stomach, because along these curvatures run the vessels which supply blood to the stomach and the main nerves of Latarjet.

As far as **duodenal ulcers** are concerned, they are preferably located on the anterior surface and their relation to the liver and pancreas explains the defensive rôle that these organs may exercise upon the course of duodenal perforation (masked perforation). The posterior relation of the first portion of the duodenum (the most frequent seat of ulcers, explainable by anatomic and physiopathologic considerations) to the hepatic artery and vena porta aggravates the prognosis of retroperitoneal perforations of the first portion of the duodenum. While discussing gastroduodenal perforations it is necessary to speak also of **recurrent perforations**, which is most convincing proof of the persistence of ulcerous disease. It may be either a reperforation of the old ulcer or of a new local lesion. The possibility of its occurrence necessitates a gastrectomy at some future date.

INTESTINAL PERFORATIONS. Here we meet perforations of different etiology which in the order of frequency are: traumatic, typhoid, neoplastic, tuberculous, luetic and simple ulcerous; the latter being most frequently observed in the small bowel. We also may add Crohn's disease or regional ileitis, which is a more anatomico-pathologic than a clinical entity, but which can manifest its presence by becoming perforated. Meckel's diverticulum, if present, may also perforate with ensuing peritonitis.

Traumatic rupture most frequently is caused by blows on the abdomen; it can also be produced by foreign bodies which entered *per vias naturales* and even by gallstones. To this group belong also perforations produced by intestinal parasites.

Traumatic perforations occur more often in the small bowel, because it is the most exposed and most superficially situated portion within the abdomen. Its considerable length and liquid contents render it prone to yield, when an outside force acts with a definite intensity over the abdominal wall. There is no direct relationship between the intensity of trauma and the gravity of the visceral lesion. In general the order of frequency of the affected viscera is the following: the middle portion of the small bowel; its superior portion; its inferior portions, the colon, duodenum, cecum and iliac sigmoid. It should not be forgotten that the mechanism of production of this type of perforation is always mechanical and may produce multiple lesions.

As far as the type of perforation is concerned it may be variable: a transverse partial section, including the mesentery, complete circular section and sometimes an incomplete rupture producing herniation of

the mucosa through the seromuscular layer. Usually the site of rupture is in the antimesenteric border of the bowel.

Perforations due to typhoid fever constitute a grave complication and occur in 3 per cent of all cases, appearing at any stage of the disease. This perforation, due to involvement of the wall by a specific process, is of obscure and dramatic symptomatology. It may occur in the course of ambulatory typhoid fever. In order to understand the mechanism of these perforations, it is necessary to recall the anatomo-pathologic type that Eberthian infection produces at the site which it involves, namely at the distal portion of the small bowel. The three phases of the affection are infiltration, ulceration and restitution. Occasionally, the third phase does not take place and in its stead perforation occurs with acute peritonitis as its sequela. Perforations of typhoid ulcers usually are observed at the vicinity of the free border of the small bowel, most frequently in the ileum, because there are situated most of Peyer's patches. The perforation may be single or multiple, of a small diameter, occasionally only of pin-point size; at times the ulcers have a crater-like appearance and in most of the cases they start from the mucosa and reach the serosa, though in exceptional cases the process goes in the opposite direction starting from the serosa to the mucosa (suppuration of the mesenteric glands in the course of typhoid fever).

Perforation of a neoplasm is rare and observed in the infiltrating type. We may observe it in the stomach, duodenum, small and large bowel, gallbladder, and so on. The anatomic type of these perforations varies, depending on the affected viscus and the nature of the neoplasm. The perforation may present a picture of "acute abdomen" and the perforation has a tortuous, irregular, excavating appearance. In the stomach the most frequent site is the pylorus because there the neoplastic process is most often encountered. Gastric carcinoma extremely rarely invades the duodenum; this can be explained by change of structure at the pyloroduodenal junction and the arrangement of the lymphatic vessels in this region. Epithelioma develops quite frequently in the large bowel; it is of the infiltrating type and occasionally perforates.

Sarcoma, if it occurs at all, affects the small bowel (primitive tumors). The prognosis in all cases of perforation of neoplasms is grave and many times perforation is the first symptom of an otherwise silent course of growth.

Tuberculous perforations usually occur in the terminal portion of the ileum at the region of Peyer's patches. They are very rare and, owing to the formation of adhesions prior to perforation, peritonitis is localized, somewhat resembling peritonitis due to neoplastic perforations. If a tuberculous lesion affects the large bowel it has a predilection for the cecum, including the appendix, the ileo-cecal junction, ascending or de-

scending colon and the sigmoid. The prognosis of intestinal perforations of tuberculous origin is always very grave.

Luetic perforations are very rare; they are observed in the tertiary period as a result of ulcerations in the cecum or colon and also as a sequela of the softening of a gumma. They may also be observed in the secondary stage. The prognosis of peritonitis produced by this type is not as grave as in the tuberculous group, because here the specific cause of the disease can be eliminated by proper treatment.

Perforation of simple ulcers occurs most frequently in the small bowel. The pathogenesis of this lesion is obscure, the symptoms prior to perforation are few and the diagnosis, therefore, is very difficult. This lesion usually is revealed after perforation has taken place. The ulcer usually is single, more frequently in men and occurs either in the first jejunal or in the last ileal loop (65 per cent). Its localization is frequently at the anti-mesenteric border of the bowel.

In this group we may also include regional ileitis and sigmoiditis, which also may perforate. At any rate, there is a group of acute peritonitis, more or less generalized, produced by perforations by no specific agents and which occur without any evident cause giving an unmistakable clinical picture without any histologic changes.

We have left to the last consideration of intestinal perforations produced by *amebiasis*. The intestinal lesion in *amebiasis* is usually observed in the large bowel (particularly in the rectum), though in exceptional cases it may be seen in the terminal ileum. In the vermiform appendix one may encounter perforative ulcerations with ensuing peritonitis.

Ordinarily the omentum adheres to the bowel by a plastic exudate, thus producing small abscesses. In some cases perforation goes not only through the serosa of the bowel, but also through the omentum, thus producing generalized peritonitis. The type of perforation under discussion occurs with some frequency during the acute or chronic course of *amebic dysentery*. It usually results from ulceration of the cecum, sigmoid, rectum or even the appendix. The prognosis of this type of peritonitis is very grave.

The group of hepato-biliary perforations brings us to study the liver abscesses perforating into the free peritoneal cavity with ensuing general peritonitis, as well as perforations of the gallbladder, which are more frequent and of varied etiology. Rupture of the hepatic abscess into the free peritoneal cavity is observed from time to time. The peritonitis produced gives a grave prognosis, because in many cases it is difficult to diagnose it and the true cause of peritonitis is found only on autopsy.

Perforation of the gallbladder is very serious, in spite of the fact that many consider the escaped bile to have bactericidal properties which

lessen danger. The gallbladder may rupture from such various causes as trauma or specific infections, such as typhoid fever. Between these two extremes we may group perforations due to stones, those that ensue in the course of acute and gangrenous cholecystitis; those which occur through torsion of the gallbladder; ruptures due to neoplastic changes in the wall and even biliary peritonitis without perforation.

Perforation of the gallbladder apart from the traumatic form, may also be of infectious or mechanical origin. The virulence of the germs in the bile determines the prognosis in peritonitis. The rupture usually is single and its seat is either in the fundus or in the neck (both these parts are most frequently exposed to pathologic changes) usually in their inferior surface. The rupture may occur as a result not only of severe, but also of mild infections of the gallbladder.

The relation of the gallbladder to the transverse colon, duodenum and pylorus explains the possibility of adhesions between these structures in the process of development of cholecystitis before rupture takes place; for this reason many times we don't observe general peritonitis after perforations. The gallbladder may perforate into the retroperitoneal cavity, though this is exceptionally rare.

Peritonitis due to perforation of the female genital tract is relatively frequent. Rupture of a tubo-ovarian abscess or of a tube only (as in pyosalpinx) is observed nearly always in patients with a previous history of gynecologic disorder. Perforation of the uterus as a result of criminal abortion is unfortunately still too frequent. It may become complicated by intestinal perforation, thus making it a double perforation of traumatic origin with a very grave prognosis. Sometimes this may occur not as a result of abortion but in the course of a diagnostic or therapeutic curettage. This, however, is rare and if done by an experienced surgeon in an aseptic environment the prognosis is not so grave. Intestinal perforation through the uterus may be single or multiple and may be complicated by injury of the mesenteric blood vessels.

Rupture of the urinary bladder, usually of traumatic origin, has to be mentioned separately because the anatomic condition of this organ in the pelvic cavity and its relation to the peritoneum will produce a different clinical picture depending on whether the rupture was intraperitoneal or extraperitoneal. In the former immediate intervention is imperative, as peritonitis starts rapidly; in the latter, the leakage of urine takes place into prevesical cellular tissue which is separated from the general peritoneal cavity. But even then, in spite of the fact that the picture does not look alarming, urinary infiltration of the cellular tissue of the pelvis with subsequent peritoneal irritation is a grave complication.

Rupture of the appendix. We are dealing now with the most frequent type of sub-diaphragmatic perforations of the digestive tract. It constitutes a grave accident in the course of acute appendicitis, and it may

occur at any moment of its course. Perforation of the appendix may be single or multiple and usually is due to deep necrotic lesions. In its pathogenesis two factors are involved, the infectious and the mechanical (the abuse of cathartics is more or less the determining factor). It is known the preponderant rôle of *b. coli* in the pathology of intestines and its association with the group of anaërobes (particularly in the course of acute appendicitis) and whose study was accomplished in the last few years showing the presence of *b. perfringens*—*clostridium welchii*—which is the most dreadful on account of the rapidity with which it penetrates the intestinal wall, *vibrio septicus* and *gas bacillus*. This study contributed greatly to lessening the gravity of the prognosis of gangrenous appendicitis with subsequent peritonitis through application of anti-anaërobic serotherapy and penicillin. In the study of appendicular perforation we should mention the rôle the Eberth bacillus may play. We believe that the appendix contains the very same conditions present in the region of the terminal ileum, namely abundant lymphoid tissue which may present the same specific anatomico-pathologic lesions, such as infiltration, ulceration and perforation. In other words, we may observe in the region of the appendix characteristic lesions of typhoid infection. We personally have observed a case of acute ulcerative appendicitis resulting in a perforation histologic study of which showed a characteristically typhoid lesion. The seat of perforation in the order of frequency is the tip, body and the appendiculo-cecal junction, the latter often occurring with perforations of the cecum. As far as the shape and size of perforation and the prognosis of peritonitis are concerned, they vary according to the general morphology of the appendix, its size, fixation, direction, virulence of the bacteria, resistance of the patient and time of surgical intervention.

Other causes of appendicular perforation are tuberculosis, actinomycosis and amebiasis, as already mentioned above.

CLINICAL STUDY

Perforative peritonitis forms an important group of acute abdominal affections and presents the surgeon a rather analogous group of symptoms, so that we are fairly justified in saying that the clinical manifestations are nearly always identical varying only in their causes.

Clinical study comprises symptomatology, differential diagnosis and treatment, the latter including also preoperative and postoperative management.

Symptomatology

Severe pain and rigidity are always initial symptoms. Later appear shock, vomitus, and so on, which supplement this disturbing picture.

Pain is the initial symptom. At times it alone enables us to make the

diagnosis. It is acute, intense, and at the beginning definitely localized. This symptom of localized pain abates after a few hours due to involvement of the peritoneum, but carefully examining the patient we still can find the place of maximum tenderness. However, this symptom may become misleading if we are first called to see the patient after he was given morphine. When we see such kind of a picture we may suspect perforation. However, that is the time to remember, that it is essential to exclude the presence of a lesion of the chest or of the nervous system such as angina pectoris, pneumothorax, pneumonia, pleurisy or tabetic gastric crisis, which may give a similar picture of "acute abdomen." We have to utilize the diagnostic procedures, such as radiography, radioscopy, urine and blood examination. Thus, presence of air in the free peritoneal cavity shows a gastro-intestinal perforation; effusions into the great peritoneal cavity are recognized by separation and arcuate arrangement of the intestinal shadow. Effusions taking place in the epiploic cavity are manifested by deformity of the greater curvature of the stomach, due to its compression. Cholecystography as well as urography offer us great help in the interpretation of acute abdominal conditions. Peritonitis appears as an immediate sequela of perforation. This is produced by effusion of irritating fluids into the peritoneal cavity. The effusion accumulates at the beginning around the perforation and later extends along the distinct compartments of the peritoneal cavity we have already discussed. The pancreatic, gastric and intestinal juices are very irritating. In order of severity of irritation they are followed by bile (choleperitonitis without visible perforation of the biliary passages) and urine. Less irritant are blood, chyle and mucus.

Contact of these irritant fluids with the peritoneum produces hyperesthesia and rigidity of the anterior abdominal wall (which is not the case with the posterior wall), clearly demonstrating peritonitis, that may become generalized. In the meantime the respiration becomes thoracic followed later by dyspnea.

If the fluid accumulates in the pelvis, Douglas' pouch becomes very tender, accompanied by contraction of the pelvic floor, which can be detected by rectal examination.

We have already described two cardinal symptoms of perforative peritonitis: pain and rigidity—the former as sequela of perforation and the latter of peritonitis.

In addition should be mentioned other symptoms: vomiting, which is neither constant nor characteristic. On the contrary, nausea is always present (Murphy). Shock, the result of intense irritation of the peritoneum, is manifested by arterial hypotension, accelerated pulse, paleness, cold sweats and restlessness. It is now accepted that in cases of visceral perforations there always exists a general vascular paralysis particularly of the capillaries and arterioles as the real cause of shock.

It has to be stated that in perforations of the viscera shock is not an initial symptoms, but appears sometimes later, coincidental with the spreading of the peritonitic exudate.

This is the general symptomatology of peritonitis produced by perforated lesions of the viscera which may be applied to all cases.

Diagnosis and Treatment

We shall now discuss the diagnosis and treatment. We refer especially to non-penetrating traumatic perforations of the viscera in the abdominal cavity; to perforations of gastroduodenal ulcers; typhoid perforations; perforations of the gallbladder and of the appendix, though in the first part of this article while discussing other causes of perforative peritonitis, we drew some conclusions of a clinical character. Accompanying the differential diagnosis we will outline the treatment in each particular case, including preoperative and postoperative management.

PERFORATIONS DUE TO TRAUMA OF THE ABDOMEN. We refer here only to perforations of hollow viscera. We already spoke of general symptoms (pain and rigidity) which, with a history of trauma, suffice to make the diagnosis and to consider surgical intervention because of the great danger of this type of trauma causing infectious peritonitis. The lower the place of rupture and the larger the amount of fluid escaped, the more rapidly peritonitis sets in. Free gas in the abdomen is common in all cases of rupture of air-containing viscera, which is possible to establish by a flat x-ray plate.

Traumatic perforation produces profound shock which manifests itself by a drop in the peripheral circulation, paleness, feeling of cold, vomitus, and the like.

Treatment of this class of perforations is surgical. The conduct to be followed is not to hesitate and wait until the trauma produces a clinical picture of "acute abdomen"; dilatory measures and waiting for acute symptoms means only to lose the opportunity to save the patient. Naturally, there may be cases in which the peritoneal syndrome appears late and silently. It is only in these cases that watchful waiting is indicated. We repeat that in the greatest majority of cases of perforation severe pain and muscular rigidity facilitate the diagnosis. Surgical intervention should be preceded by preoperative management, directed to combat the shock and consists of administering plasma, normal salt solution, glucose, adrenaline, caffeine. Blood transfusion may be employed before or after operation according to circumstances.

GASTRODUODENAL PERFORATIONS. We will discuss especially those produced by ulcerative lesions. From our personal statistics, comprising a fairly large number, we may draw the following conclusion. Three factors are worth consideration, namely age, sex and the presence of gastric symptoms previous to the accident. As regards sex, we had not a single

case which occurred in a woman, and this concurs with the statistics of others. With respect to age, except the rare cases of perforation at the extremes of life (before 15 and after 60 years), I have to say that this accident occurred at any age.

As far as presence of gastric disturbances prior to perforation is concerned, I have to say that I always found a history of digestive disturbance, such as dyspepsia, hyperchlorhydria and so on, more or less accentuated. I did not observe it in duodenal lesions, in which case, having a picture of perforation in the upper abdomen, absence of preceding gastric symptoms may serve as a possibility of a perforated duodenal ulcer.

As far as the diagnosis of perforation is concerned, we think that with careful observation of each case, the two cardinal symptoms pronounced, such as severe pain, localized first in the epigastric region, and later spreading into the right iliac fossa and at times into the entire lower portion of the abdomen and followed by muscular rigidity, suffice to make the diagnosis.

Disappearance of the hepatic dullness I have observed frequently. It has its importance. The differential diagnosis from acute perforative cholecystitis is based on the site of the pain, because in the latter it is localized in the right hypochondrium, with radiation into the shoulder of the same side and with maximum rigidity at the same place.

The differential diagnosis from hemorrhagic pancreatitis is more difficult, rendering it necessary to resort from time to time to determination of the diastase in the urine and even to x-ray. If a few hours elapsed since the accident, the differential diagnosis from acute perforated appendicitis may become difficult. We can say that every surgeon made this error, because escape of the contents from the perforated ulcer into the right iliac fossa perfectly simulated a perforated appendix. Therefore, one should not forget to carefully ask the patient to determine the site of the greatest intensity of the pain, because in ulcer this is observed at the region of the epigastrium.

The treatment is surgical. But what kind of intervention should be employed? This is a problem which even now is not settled by surgeons. Three methods are employed: first, simple suture or invagination with or without gastroenterostomy; second, excision of the ulcer or its cauterization followed by closure; and third, gastrectomy. One thing is accepted by every one and that is early intervention.

We are in favor of simple suture or invagination followed by reinforcing with omentum, because we consider that for a patient who sustained a gastro-intestinal perforation all that is necessary, all that is required, is to close the opening, which permits the escape of gastroduodenal contents and brings about fatal peritonitis. Suture should be done in two layers, followed by epiploplasty (Fig. 244). In

cases in which closure is impossible on account of local conditions of the tissues, we are content with simple closure with omentum. Gastrectomy in our opinion is dangerous for such cases not only on account of the general condition of the patient, but also because the operative field is

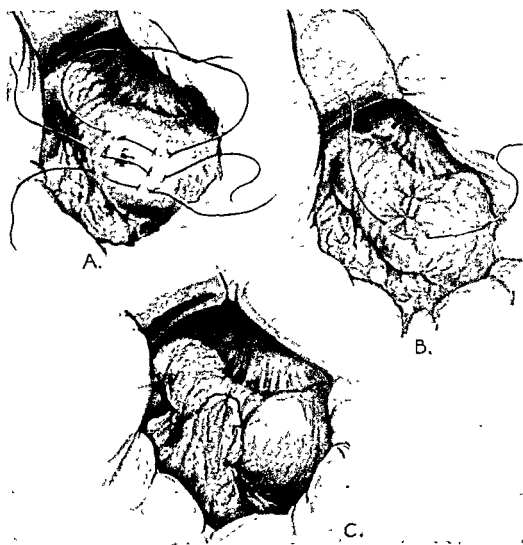


FIG. 244. Repair of a perforated ulcer. A. Simple occlusion; first layer of sutures. B. Simple occlusion; second layer of sutures. C. Simple occlusion reinforced by omentopexy.

contaminated even if the operation is done within the first six hours. By the same token we should be not eager to perform gastroenterostomy, unless it becomes a necessity. We think, therefore, that the infectious condition due to perforation is a contraindication to any prolonged operation. There is a general opinion to which we also subscribe, that acute gastroduodenal perforations after six hours have a grave prognostic outlook. As far as drainage is concerned we always place a small cigarette

drain in the vicinity of the lesion and in exceptional cases resort to suprapubic drainage.

As preoperative treatment we employ the same methods as in traumatic ruptures. Following the operation we administer glucose, cardiac stimulants and in some cases antiperitonitic serum—all with good results. Equally, blood transfusion is indicated. Very often we place our patients in Fowler's position. It is necessary to mention that this group of peritonitis may be accompanied by postoperative complications of a cardio-pulmonary nature, by leukocytosis and positive intradermal reactions for the bacteria of the entero-streptococcic group.

TYPHOID PERFORATIONS. The diagnosis of intestinal perforation in the course of typhoid fever is occasionally difficult. Generally we can say, that the two cardinal symptoms of gastroduodenal perforation are present also here, though not with the same clarity and exactness. Therefore, in some cases the diagnosis is clear, in others we arrive at it only when there is general peritonitis and when surgical intervention is already late.

For this reason, typhoid fever patients should be watched by a surgeon as well as by an internist. Typhoid fever should be looked upon as a medico-surgical entity on account of its irregular course and possible complications. Therefore, in cases with a highly toxic course we have carefully to watch symptoms of pain and circumscribed rigidity, because these two symptoms are always early ones and may signify that perforation may take place at any moment.

The symptom of sudden drop of temperature does not have the significance that some liked to assign to it. To pain and rigidity are added other symptoms which reveal peritonitis.

We should not forget the possibility of another complication during the course of typhoid which develops in the abdomen and may be confused with intestinal perforation of ileum. I refer to perforated typhoid ulcer of the appendix. Other acute abdominal conditions which may appear in the course of typhoid are acute pancreatitis, acute intestinal obstruction, perforative cholecystitis, mesenteric thrombosis and intestinal hemorrhage. *We should not forget that these conditions whenever suspected should be treated surgically.* Accordingly, even a diagnostic error in such cases would be salutary for the patient, because early laparotomy is the most urgent indication in such cases.

As far as treatment is concerned, the preoperative management is the same as that for other causes of perforation. The operative measures consist of simple suture; or suture following previous excision; or of closure with omentum or adjoining mesentery; or of resection of the bowel, or of enterostomy and, finally, exteriorization of the loop.

In our personal statistic we had seven cases of perforation of the ileum and one of the appendix. Except the last one, which was removed

and then only diagnosed microscopically as perforated typhoid ulcer of the appendix by Professor Hoffman (to whom we are indebted for the anatomico-pathologic study of all our cases of perforation of the gastrointestinal tract), in all our cases we employed simple suture in two layers, with excellent results. Therefore, we consider this method as very simple and rational, remaining loyal to certain fundamental principles in urgent surgery, to wit: the best methods are the simple ones; to adjust the operative procedure to the condition of the patient; to search for the cause which produced the condition; to traumatize as little as possible so as not to aggravate already existing shock. In other words, all this can be condensed in one phrase enunciated by Murphy, particularly for cases of "acute abdomen"—"to get in quick and get out still quicker."

Some surgeons give preference to enterostomy, which, according to Witzel's method, is simple and can be done rapidly. In these cases we give the patients adequate preoperative and postoperative treatment, not neglecting anything in treatment of the original disease.

The prognosis of this type of perforation is very grave; it is not the same whether perforation takes place in the first days of the disease or when the sickness reaches its greatest gravity, as when it occurs in the third week of typhoid fever, because in the first case the patient still has enough defensive power, reacts better, which in its turn is demonstrated by the fact that perforation is manifested by symptoms usual for such a condition, namely a violent, acute course and easy diagnosis. However, when the patient is already exhausted, depressed, dehydrated, when his defensive powers are dwindled and perforation then takes place, the symptoms are obscure and the prognosis is dubious. The clinical form of the infection will influence the prognosis of perforation. Multiple perforations, which occasionally may pass unnoticed, as well as the possibility of appearance of new ones greatly increase the gravity of the condition.

PERITONITIS DUE TO PERFORATION OF THE GALLBLADDER. It may be observed, as we already mentioned, in the course of cholecystitis or cholelithiasis, acute catarrhal or gangrenous inflammation; it occurs at any age and preferably in women. Nearly always there is a history pointing to disease of the gallbladder and occasionally of acid dyspepsia which facilitates the diagnosis. Perforation in these patients may be confused with acute biliary colic. However, in perforation the pain is greater, being first localized in the region of the gallbladder and later generalized, predominating in the right iliac fossa. That is the reason why this affection is often mistaken for acute perforated appendicitis. Nevertheless, taking carefully the previous history of the patients pointing to the presence of stones, the intensity of the symptoms, particularly pain and its localization, will help to establish the differential diag-

nosis of this type of peritonitis from other types encountered in acute abdominal conditions. The prognosis of this type of peritonitis is very grave and depends chiefly on early intervention.

Treatment is surgical and we prefer cholecystectomy as the operation of choice. However, exceptional circumstances may force us to employ simple cholecystostomy. As far as preoperative and postoperative treatment is concerned, it is the same as for perforation due to other causes. Glucose with or without insulin, liver preparations and so on find here their natural indication.

BILIARY PERITONITIS WITHOUT ACTUAL GALLBLADDER PERFORATION has been interpreted differently. Necrosis of the gallbladder wall has been attributed by some to a return flow of pancreatic juice or duodenal contents through the ampulla of Vater. In the first case there will be spasm of Oddi's sphincter, in the second case relaxation of the same.

This condition starts with a picture of acute cholecystitis and when the pain reaches the right iliac fossa, one may mistake it for acute appendicitis. One does not think of biliary peritonitis due to perforation because in this case there is absent the initial violent attack of pain, because the irritation of the peritoneum by bile is progressing slowly. Nevertheless, careful observation of each case, persistence of the symptoms and their accentuation, the toxic state of the patient with a tendency of collapse, will help us greatly to decide on only one type of treatment and that is surgical. Fortunately, this type of lesion occurs exceptionally rarely. With reference as to how to proceed, the statistics show that cholecystectomy is the best method. While treating biliary peritonitis one should not forget that it may be due to perforation of the common bile duct, though this is extremely rare. If such is the case, it is due to perforation by a stone and the symptoms are identical with those of perforated gallbladder. The urgent intervention consists of extracting the stone or stones and drainage of the duct by a T-tube or placing a catheter into the upper part of the same duct.

PERFORATED APPENDICITIS. This is the most common type of perforation of the viscera and constitutes 95 per cent of all perforations. Deaver said: "Acute abdomen in the absence of an abdominal scar should make us think first, last and always of appendicitis." There is a great frequency of perforated appendicitis in Cuba, particularly among the Spanish element. Its frequency is so great that we had an opportunity to operate in a single day five such cases. As far as age and sex are concerned we observe the greatest number between 20 and 30 years and especially in men.

We also observed nearly always that the patients are constipated and many times we found in the lumen of the appendix parasites, particularly trichocephalus. This was verified by systematic anatomico-patho-

logic examinations. In exceptional cases we also had amoeba histolytica, occasionally mixed forms of amoebae are encountered.

The diagnosis of perforated appendicitis requires first the establishment of the diagnosis of appendicitis and then of perforation. To prove the first point is occasionally difficult. So far as the second problem is concerned, it is easier but of lesser importance, because all cases of acute appendicitis require immediate surgical intervention. Under such consideration it is of no importance to try to diagnose precisely whether or not there is a perforation.

There are many conditions which may obscure the diagnosis of acute appendicitis.

Suffice it to say, that it will be necessary to differentiate it from all lesions of an "acute abdomen": acute cholecystitis, acute gastro-intestinal affections, intestinal obstruction, Meckel's diverticulum, typhoid perforations, and from pneumonia, pleurisy, disorders of the genito-urinary tract (uterus, adnexa, pelvic cellulitis, etc.) and extrauterine pregnancy.

As far as the diagnosis of perforated appendix is concerned, this manifests itself either as a localized type or one perforating into the free peritoneal cavity. It manifests itself by violent pain, which later abates, and an accelerated pulse. Frequently the patient has a severe chill, acute pain and generalized rigidity of the abdominal wall with the maximum intensity at the right iliac fossa. In other cases perforation of the appendix is manifested from the beginning by symptoms of diffuse peritonitis.

The prognosis of perforated appendicitis is less grave than peritonitis of other origin. Here, as in all other cases, the time element is of greatest importance. The prognosis also varies, depending on whether perforation was localized or ruptured into the free peritoneal cavity, the position and direction of the appendix, and so on.

Treatment should be nothing else but surgical. The preoperative measures consist of increasing the strength of the patient, treating the shock, and so on, by injections of fluids, simple or to which is added adrenalin, of cardiac stimulants, of blood transfusion in small quantities and even of antiperitoneal serum. Sulfa drugs and penicillin are of greatest benefit. Removal of the appendix is an ideal procedure and we say it because there are cases, though exceptionally rare, in which we limit our action to drainage of the region, especially in circumscribed peritonitis. As soon as the appendix is removed we place a cellophane drain; at times we employ a Mikulicz tampon which saved us many patients. We also employ systematically polyvalent antiperitonitic serum parenterally and into the abdominal cavity through a drainage opening and we have to confess that our statistics in perforated appendicitis with following peri-

tonitis are really gratifying. With the use of the serum the temperature drops immediately; intoxication clears up. With the serum we use a great amount of normal salt solution and glucose, Fowler's position, cardiac stimulants and blood transfusions.

As a supplement to postoperative treatment in peritonitis due to perforation in general we have to mention the great help afforded by continuous drainage of duodenal contents by Wangensteen's method. We always employ it when obstructive symptoms are present. It is used for hours or days and affords prophylactic and curative treatment of paresis of the intestinal wall due to peritonitis. We also use morphin in small repeated doses, which increases the intestinal tonus and prevents its overdistention. The use of hypertonic solutions intravenously is recommended.

As a general conclusion we may say that from the clinical study of peritonitis due to perforation we can clearly see that it should be included into the group of lesions of "acute abdomen." Taking into account the general symptomatology we have described with the two cardinal symptoms—pain and rigidity—it is essential to establish the differential diagnosis. We wish to say that it is necessary to rely upon clinical signs and not to delay any time for laboratory tests which are only auxiliary diagnostic means.

As far as treatment of peritonitis due to perforation is concerned we can say that the method of actual operation depends on the type of visceral perforation and on circumstances. In all cases we follow the preoperative and postoperative management according to the enunciated rules and, finally, we plead for early intervention as the only means for reducing, as far as possible, the somber prognosis of this type of lesions.

Chapter XVIII

Non-perforative Peritonitis

By JULIUS L. SPIVACK

In most cases peritonitis is due to contamination from a ruptured viscus, and has been discussed in the chapters on perforative peritonitis and surgery of the gall bladder. In some cases it is a late sequel of intestinal obstruction or of mesenteric thrombosis. However, in some rare cases it occurs through hematogenous or lymphogenous dissemination or spreading by continuity. In this chapter we shall discuss peritonitis of non-perforating origin.

Peritonitis is inflammation of peritoneum, which is characterized by various local and general manifestations. Depending on the extent of the inflammation peritonitis may be localized or generalized; on the degree of inflammation—acute or chronic; on the causative factor—bacterial or chemical; and on the source of infection—primary or secondary.

Peritonitis usually presents three stages:

The *first* stage lasts a few to twenty-four hours. At this stage there is a moderate elevation of the temperature of one to two degrees, moderate increase in the pulse rate up to 100, moderate pain, tenderness and muscular rigidity. There is moderate leukocytosis, slight drop in the systolic pressure, while the diastolic remains normal. This stage is very often masked by symptoms of the disease which produced peritonitis and, therefore, is easily overlooked. If the source of inflammation has been checked by surgical intervention or spontaneously the disease may terminate in recovery at this stage. If, however, the growth of the microorganisms continues and the defense mechanism is unable to check it, the second stage takes place.

Second stage. This lasts from twelve to seventy-two hours, depending on the virulence of the bacteria and the effectiveness of the bodily defensive mechanism. It is characterized by multiplication of bacteria, stagnation of blood in the capillaries of the peritoneum with ensuing thrombosis. Peritoneal exudate is seen in a moderate amount.

Clinically, the symptoms are manifested by nausea, vomiting, increased abdominal pain, muscular tenderness and rigidity. The temperature reaches up to 102°F., the pulse is disproportionately rapid, the respirations become shallow. Albuminuria is present, some red blood cells are in the urine and the leukocyte count varies between 15,000 to 30,000. Even in this stage recovery may take place after appropriate surgical or chemotherapeutic measures. If, however, no resolution occurs the peritonitis enters into the third or final stage.

Third stage. Pathologically it is characterized by an abundant exudate in which is found an enormous amount of bacteria; the blood capillaries are markedly dilated and thrombosed.

Clinically, the patient is toxic; the pulse is fast and irregular; the respiration is fast, shallow and labored. Oliguria or even anuria may take place. The temperature may rise to 105°–106°F.; the pulse rate to 160 and beyond so that it can no longer be counted. The systolic blood pressure drops, while diastolic is lowered very little, if at all. This stage lasts from twelve to seventy-two hours. Death usually is due to toxemia, myocardial insufficiency, anuria or cerebral edema.

Treatment of peritoneal infection can be grouped into two categories:

1. Destruction of the injurious agents by chemical agents, or withdrawal of these injurious materials by surgical means, drainage and the like;
2. Enhancement of the natural body resistance by the introduction of blood, plasma, glucose, normal salt solution.

We have presented a general outline of the symptoms and the treatment of peritonitis in the acute stage irrespective of the cause. However, there are rare types of peritonitis, which may vary in details from this outline both in their symptomatology and treatment.

Bile Peritonitis

This has been discussed in the chapter on "Urgent Surgery of the Liver."

Tuberculous Peritonitis

ETIOLOGY. *Incidence.* Tuberculous peritonitis with clinical manifestations occurs in about 0.5 per cent of all individuals afflicted with tuberculosis. However, on autopsies signs of tuberculous peritonitis are found in about four per cent of all tuberculous patients. In other words, in many cases there is tubercular involvement of the peritoneum which does not manifest any clinical symptoms and remains unrecognized. In most of the cases tuberculous peritonitis is secondary to some primary focus of infection; it is extremely rare as a truly primary infection.

AGE. It occurs most frequently in the third decade.

SEX. Clinically there is a female preponderance over the male, the ratio being 4:1. However, on autopsy the reverse is true; there is a male preponderance of 3:1. This can probably be explained by the fact, that constant low abdominal pain in females is a cause for surgical intervention, while low abdominal pain in the male is mostly treated medically.

RACE. Negroes are more often affected than whites.

PATHOLOGY. Tuberculous peritonitis as a rule is secondary to a pathologic focus in some other viscus, particularly the lung or intestine,

A primary focus in the peritoneum is extremely rare. It may be acute or chronic and in either case localized or diffuse.

SYMPTOMATOLOGY. The onset may be insidious or sudden. It is manifested by *pain* which is the earliest symptom. This pain may be severe or mild, generalized or localized. In the latter case it may be in the lower or the upper abdomen. If in the lower abdomen it may be suprapubic, in the right or in the left lower quadrant. This pain may last for months and has no relation to the intake of food.

Abdominal distention appears next in frequency. It is due to ascites or gas. The ascitic fluid may be clear, or cloudy, chylous or hemorrhagic. The temperature is moderately elevated ranging between 99° and 101°F. The pulse is moderately accelerated, corresponding to the rise in temperature.

Tenderness is present; it may be diffuse or localized, depending on the pathologic process.

Abdominal masses are occasionally present. They consist of agglutinated loops of bowel, or of rolled omentum.

TREATMENT varies depending on the character of each individual case. In primary tuberculosis with ascites, laparotomy is still the favorite method of treatment. Whatever might be the explanation, it is an undisputed fact, that a mere laparotomy gives marked relief if not a cure in many cases. Some authors advise as a therapeutic measure the production of pneumoperitoneum by injecting nitrogen or oxygen. However, the therapeutic value of pneumoperitoneum is questionable. Other surgeons advise irradiation by giving moderate dose of x-ray over the abdomen for a prolonged period of time; however, the results are questionable. If the peritonitis is secondary to some primary focus, the treatment of the latter is the most important measure. In tuberculous salpingitis, removal of the fallopian tubes is advisable. In pulmonary conditions the latter are treated by measures customarily used in the management of lung tuberculosis depending on the pathologic process present in the lung.

PROGNOSIS of tuberculous peritonitis depends on the character of concomitant pathology as well as on the extent of the peritoneal infection and the degree of acquired immunity.

Active pulmonary tuberculosis associated with tuberculous peritonitis gives a high mortality, ranging from 50 to 75 per cent. Gastro-intestinal tuberculosis associated with tuberculous peritonitis also shows a high mortality rate.

Tuberculous peritonitis associated with tuberculous salpingitis has a good prognostic outlook, giving a mortality of about 15 per cent, if the fallopian tubes are removed.

Primary uncomplicated tuberculous peritonitis offers the best prognostic outlook.

Gonococcic Peritonitis

It occurs in adults as well as in children. It is rather a very infrequent occurrence, being present in about 2 per cent of acute vulvovaginitis in children, and more often in adults.

PATHOLOGY. Gonococcic peritonitis may be either in an active or in inactive, adhesive stage. In the first case it may be generalized, affecting the entire abdominal cavity, or localized, involving either the pelvic or the upper abdominal portion, chiefly as perihepatitis. In the healed or adhesive form it produces a great amount of adhesions. Most frequently gonococcal peritonitis starts in the pelvic cavity, particularly in women. Gonococcal peritonitis is usually secondary, the primary focus being in the external genitalia from which it spreads either by a direct route through the fallopian tube or by lymphatic extension, not involving the tubes. Finally, it may affect the peritoneum hematogenically.

SYMPTOMATOLOGY. The onset is sudden with acute abdominal pain, which may be diffuse or localized in the lower abdomen; the temperature is high, reaching 105°F., with the pulse disproportionately high; the leucocyte count varies from 20,000 to 60,000 per cub. millimeter.

There is moderate tenderness, rigidity and abdominal distention. Up to the advent of the sulfa drugs the disease lasted from a few days to several weeks and in the fulminating types the patients died within twenty-four hours.

PROGNOSIS. With the advent of the sulfa drugs the prognostic outlook changed completely. Whereas before the use of sulfa drugs the mortality in the young was about 25 per cent and considerably lower in adults, today the disease can be aborted. Many cases are now reported in which the acute symptoms subside within two or three days after the administration of sulfa drugs, though in some cases the patients are resistant to this drug.

TREATMENT chiefly consists of the administration of sulfathiazole and sulfadiazine orally or intravenously.

Puerperal Peritonitis

Puerperal peritonitis follows parturition or abortion. The infection may be introduced by the hands of the obstetrician or midwife or by instruments used for delivery or curettage. Peritonitis may appear in a few days in early cases and in a few weeks in delayed cases. In early development the source of infection is usually extraneous, from instruments or from the hands of the surgeon, or from the external genitalia; in delayed cases the source is some dormant infectious process in the body, such as chronic salpingitis.

SYMPTOMATOLOGY. The onset is sudden with chills, fever, reaching 103°F., acceleration of the pulse up to 160, severe abdominal pain, par-

ticularly in the lower portion. There is abdominal tenderness, moderate rigidity and moderate distention. The leukocyte count ranges between 12,000 and 35,000.

PROGNOSIS. Before the advent of sulfa drugs the mortality was about 50 per cent. Administration of the sulfa drugs has greatly improved prognostic outlook, provided the diagnosis is made early because it is now fairly well established, that chemotherapy with sulfa drugs considerably reduces the mortality of peritonitis, if given in the first stage or in the early part of the second stage. If, however, the administration of the sulfa drugs is started only in the third stage little can be expected from chemotherapy.

Syphilitic Peritonitis

This is a very rare complication of tertiary or of hereditary lues. It is encountered either as a generalized or a localized form. The latter is more frequent and is produced by extension of the luetic process from the liver, spleen, or bowel into the adjacent peritoneum, thus producing perihepatitis, perisplenitis or perienteritis. The generalized form of luetic peritonitis is produced either by rupture of a gumma or less frequently hematogenously.

SYMPTOMATOLOGY. The onset is sudden, with severe abdominal pain, generalized or confined to the right upper quadrant in case of perihepatitis, or to the left upper quadrant in case of perisplenitis. This pain is of a varying degree of intensity. There is tenderness, rigidity, accumulation of peritoneal fluid ranging from a few hundred to several thousand cubic centimeters of yellow-gray fluid. The temperature fluctuates between 99° and 103°F. The pulse is correspondingly accelerated. Leukocytes range from 10,000 to 15,000 per cub. millimeter. Serological tests for lues are positive. The course of disease may be prolonged in many cases resembling that of tuberculous peritonitis. Treatment is antiluetic.

Rheumatic Peritonitis

Rheumatic peritonitis is an extremely rare complication. It is even difficult to determine the ratio of its occurrence as a complication of general rheumatic fever because there are many statistical reports comprising thousands of cases in which not a single one of rheumatic peritonitis has been reported.

SYMPTOMATOLOGY. The symptoms related to peritonitis may in some cases precede any other manifestation of rheumatism. In other cases they appear after rheumatic fever has presented different manifestations. The peritoneal symptoms are pain in the abdomen, generalized or localized, tenderness, rigidity and abdominal distention due to accumulation of a large amount of fluid.

The elevation of temperature, accelerated pulse and leukocytosis may be manifestations either of peritoneal involvement or of rheumatic disease affecting other viscera.

The prognosis depends on the extent of rheumatic involvement of other viscera, as rheumatic peritonitis in itself is not dangerous to life.

The treatment consists of the administration of a large dose of salicylates. A large effusion requires paracentesis.

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